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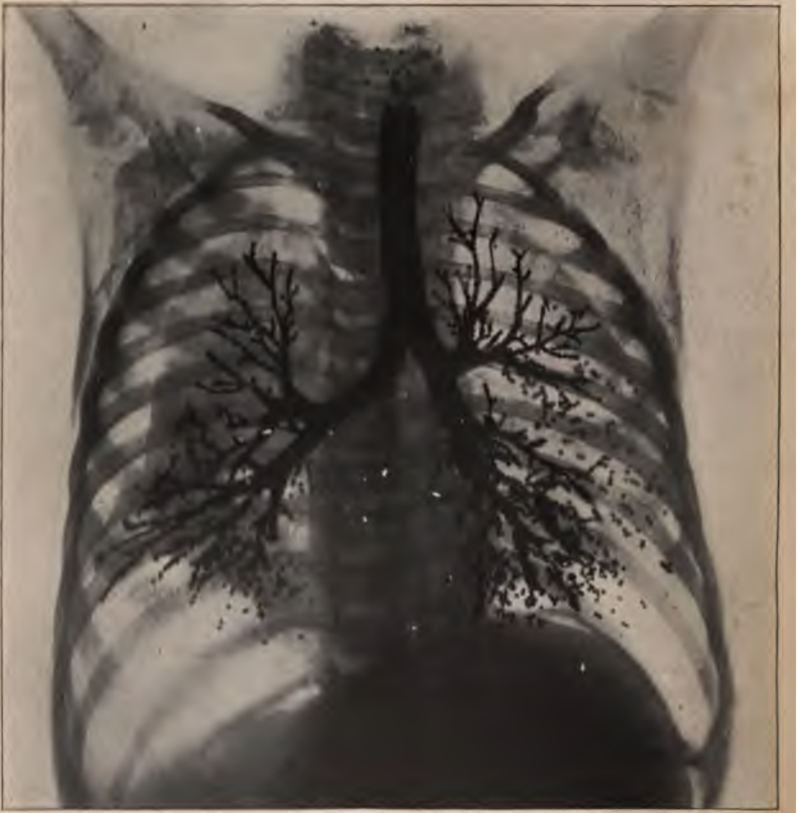

L

Radiograph of Bronchial Tree (child). Bronchi filled with Shot No. 10. Diameter, .038 in.

PHYSICAL DIAGNOSIS

DISEASES OF THE THORACIC AND ABDOMINAL ORGANS.

A Manual for Students and Physicians.

BY

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ILLUSTRATED WITH 74 ENGRAVINGS AND 12 MONOCHROME PLATES

LEA BROTHERS & CO.
PHILADELPHIA AND NEW YORK
1902

TO MY FORMER STUDENTS,

WHOSE INSISTENT

"WHY?"

FURNISHED THE INCENTIVE

FOR THIS WORK.

PREFACE

The great number of works on Physical Diagnosis deterred me for a long time from writing another, and it was only at the urgent request of a number of my former students that I undertook to put in permanent form the methods that I have used for a number of years in teaching.

Especial emphasis has been laid on the altered anatomy of the organs under examination, and its relation to the physical signs. The respiratory and cardiac sounds, their production and their modifications, both normal and pathological, have been discussed more fully than in most books of like scope, as experience has shown that unless the student clearly understands how these sounds are produced the tendency is to regard each variation from the normal as pathognomonic of special disease, rather than dependent on changes in structure or function which may be present in conditions not necessarily pathologic.

In this way only can diagnostic values be estimated, and the range and limitations of Physical Diagnosis be understood.

Physical Diagnosis is now taught at an early period in the curricula of the medical schools, and this circumstance has made it necessary to give a brief account of the morbid changes in different organs, and of their secondary effects, both immediate and remote.

In the preparation of this book I have gleaned freely from other works on Physical Diagnosis, and from articles in the medical journals.

I am indebted to Prof. C. G. Coakley and to my associate, Dr. S. A. Brown, for many valuable suggestions; to Dr. Henry J. Prentiss, director of practical anatomy, for the chapter on Regional Anatomy, the production of many of the illustrations and the preparation of the material for the radiographs of the bronchi; to Mr. Eugene W. Caldwell, director of the Edward N. Gibbs Memorial X-Ray Laboratory, for the radiographs and for suggestions in chapter on Examination with X Ray; and to Dr. Titus M. Coan for valuable assistance while the work was going through the press.

E. Le F.

52 West Fifty-sixth St., New York.
September, 1902.

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PHYSICAL DIAGNOSIS.

PART I.

ANATOMICAL.

CHAPTER I.

REGIONAL ANATOMY.

THE following anatomical facts will serve as an aid to the location of thoracic and abdominal viscera.

The trunk is divided by the diaphragm, or midriff, into two great cavities, the thorax and abdomen.

The diaphragm is a muscular, tendinous structure, attached by its circumference to the ensiform cartilage, six or seven lower costal cartilages, the external and internal arched ligaments, and upper two or three lumbar vertebræ by two crura. It is inserted into a central trifoil tendon, and points convexly upward; therefore increasing the vertical diameter of the abdomen at the expense of that of the thorax. In moderate expiration this convexity reaches to the level of the fourth intercostal space on the right, and to the fifth on the left. This gives a general landmark for the base of the lungs and heart apex above, the upper limits of the liver and stomach below.

The limits of the thorax extend from the origin of the diaphragm to the apices of the lungs, at the level of the first dorsal vertebra behind, or one-half to one inch above the clavicles, but deeply placed behind the sterno-cleido-mastoid muscles and scalenus anticus muscles.

THE SUPERFICIAL LIMITS OF THE ABDOMEN.

Above they are formed by the attachments of the diaphragm to the costal arches, etc.; below by the pubic bones, Poupart's ligaments and iliac crests.

TRUE LIMITS OF THE ABDOMEN.

The true limit above is the vault of the diaphragm; that below is the floor of the pelvis, *i. e.,* levatores ani, etc.

THORAX.

The thorax is a conical-shaped structure, whose walls are: sternum and costal cartilages, anteriorly; ribs, laterally and posteriorly, and the twelve dorsal vertebræ in the posterior median line. Its floor is the diaphragm; its apex is determined by the apices of the lungs, which are limited by the scalenus anticus muscles, beneath which the lungs project into the neck. The cavity is flattened from before backward, and above is reniform in shape on cross section, due to the marked anterior projection of the bodies of the dorsal vertebræ.

The sternal wall is obliquely placed, being nearer the posterior thoracic wall at its upper than lower limit.

It begins at the episternal notch, opposite the second dorsal vertebra, and terminates at the ensiform cartilage, opposite the ninth dorsal vertebra.

The vertebral wall begins at the first dorsal vertebra and ends at the twelfth; therefore the thoracic cavity is longer behind than in front, sloping downward and backward.

Surface Markings of Thorax.

Anteriorly, Median Line.—Above is the episternal notch or jugular fossa, located between the sternal heads of the sterno-cleido-mastoid muscles and above the first piece of the sternum. Passing down, we come to the sternal groove or furrow. Its floor is the sternum, subcutaneously placed, its lateral boundaries the sternal origins of the great pectoral muscles. Running the finger down this furrow, it comes to a marked ridge, the sternal angle, or angle of Ludovici. It is formed by the union of the first and second pieces of the sternum, and is opposite the articulation of the second costal cartilages —a valuable landmark in the counting of ribs. At the inferior extremity of the second piece of the sternum or gladiolus is located the infrasternal fossa (epigastric fossa or scrobiculus cordis). It is well marked, because of the prominent sternal articulations of the seventh costal cartilages and depression of the third piece of the sternum (ensiform or xiphoid cartilage).

Laterally.—Passing from the first piece of the sternum almost horizontally outward and backward are the two clavicles, which overlie the first ribs. At the junction of the convex inner two-thirds with the concave flat outer one-third of the clavicles are felt the coracoid processes, projecting forward one-half to three-quarters of an inch from beneath the clavicles. Directly external to the coracoid processes we come to the upper extremities of the humeri, their lesser tuberosity and bicipital ridge being directly under the finger.

Above the clavicles are the supraclavicular fossæ, into which the apices of the lungs project, but deeply placed beneath the sterno-cleido-mastoid and scalenus anticus muscles. Normally, these fossæ are shallow.

Below the clavicles are the faintly marked infraclavicular fossæ. Below the second ribs are the second intercostal spaces; the right being called the aortic interspace, the left the pulmonic.

The nipples in the male and young females are usually found in the fourth intercostal spaces, about four inches from the median line.

Passing downward and outward from the junction of the gladiolus and ensiform cartilages are the deviations of the cartilages of the seventh, eighth, ninth and tenth ribs, forming the subcostal angle, which is more acute in the case of a long, narrow thorax. Running the finger down either side of this angle, we discover that the eighth costal cartilage is firmly attached to the seventh, while the ninth and tenth are freely movable, having a loose fibrous attachment to the cartilage above, instead of articular facets. The eleventh and twelfth ribs are felt below and behind the tenth.

With the arms raised, the hands resting on the head, the axillary regions are exposed. The anterior and posterior borders of which, when continued vertically downward, form the anterior and posterior axillary lines; the vertical line midway placed is the mid-axillary line. It is in this region that the lungs are nearest the examiner.

Posteriorly.—In the median line is the vertebral furrow, at the bottom of which can be felt the vertebral spines. This furrow is more marked in the cervical and lumbar regions than in the dorsal and sacral regions.

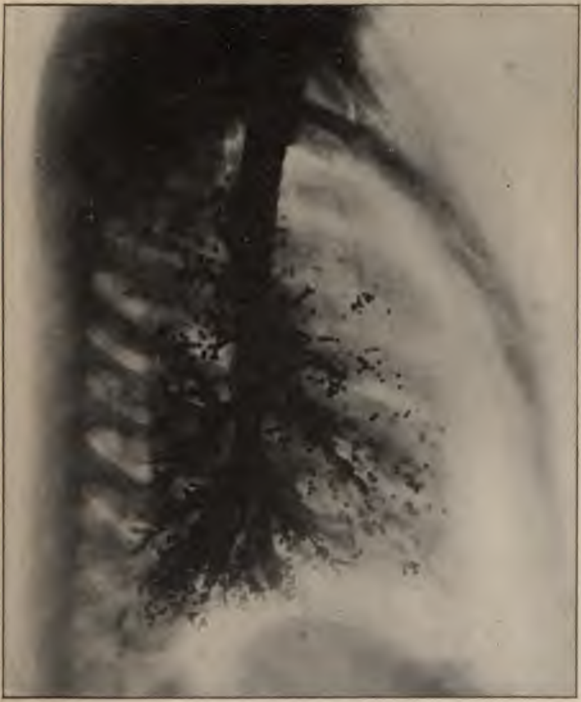
Though the seventh cervical vertebra is called the vertebra prominens, the first dorsal spine is more prominent, projecting posteriorly perhaps one-fourth of an inch further than the seventh cervical spine. Laterally, we come to the scapulæ, located between the second and seventh dorsal vertebræ, thus simplfying the location of the ribs posteriorly. The spines of the scapulæ meet their vertebral borders usually opposite the fourth dorsal vertebra.

The vertebral borders of the scapula are located about one and one-half to two inches from the median line, but deviating slightly towards the inferior angles. The interspace is called the infrascapula region. The region above each scapula spine is called the suprascapula region; below, the infrascapula region.

Contents of Thorax.

Trachea, two bronchi, two lungs, two pleura, heart, with its pericardium, great vessels, œsophagus and thoracic duct.

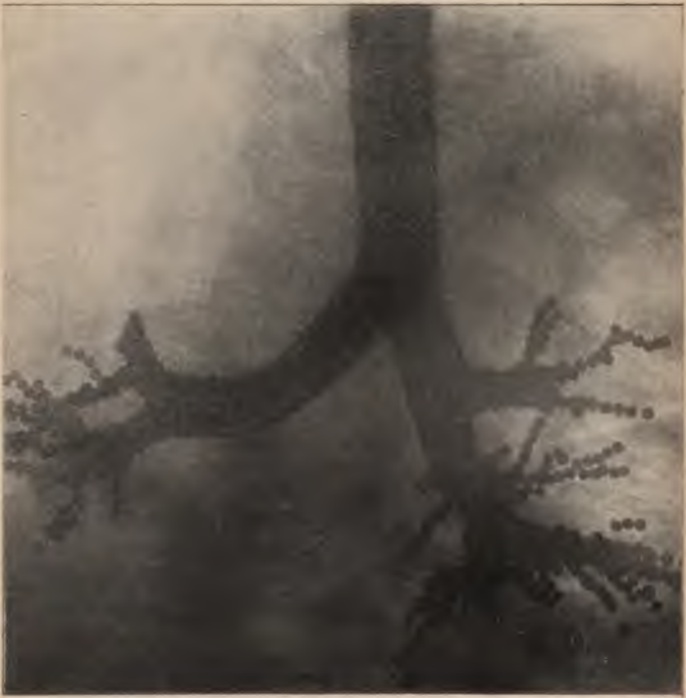
FIG. 1.



Bronchi (child) filled with shot, No. 10. Diameter, .038 inch. Lateral view.

Trachea.—Four to four and one-half inches in length. Runs slightly obliquely from above downward and backward a little to the right of the median line. It begins at the cricoid cartilage of the larynx opposite the sixth cervical vertebra and terminates at its bifurcation into two bronchi opposite the fourth dorsal vertebra, or in the plane passing horizontally from the junction of maubrium and gladiolus to the body of the fourth dorsal vertebra. (Plate II, and Fig. 1.)

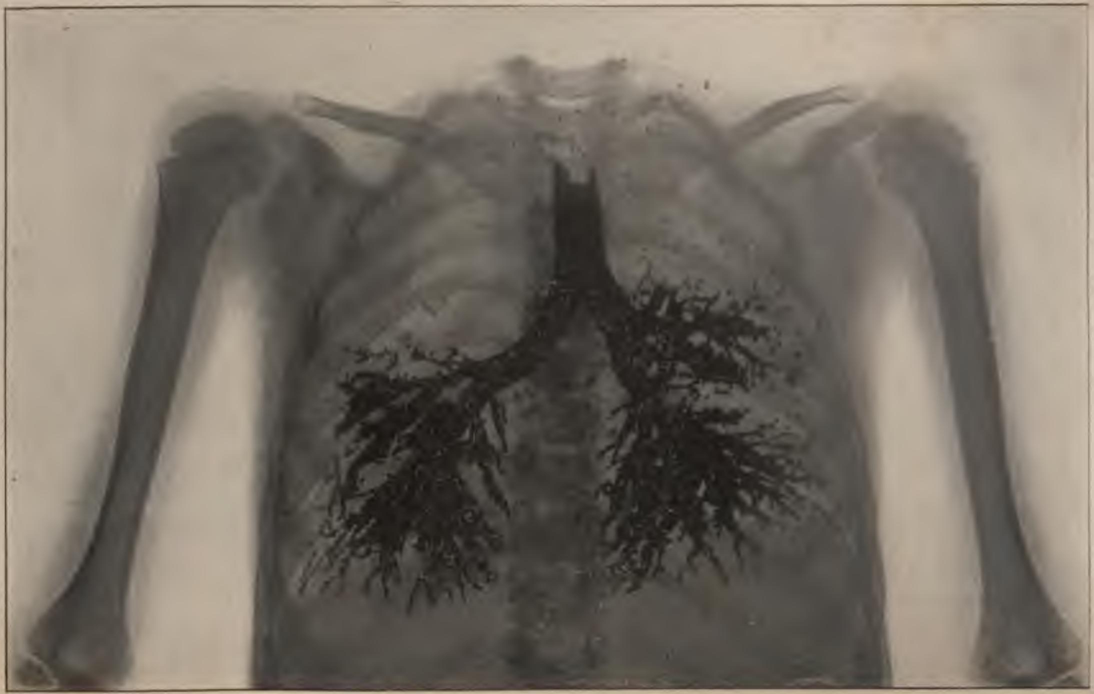
FIG. 2.



Bronchi (adult) filled with shot, No. 4. Diameter, .110 inch.

Bronchi. RIGHT AND LEFT.—Two to two and one-half inches long from the bifurcation to where they

PLATE II.



Bronchi (child) filled with Shot No. 10. Diameter, .038 in.

break up into bronchia at the hilum of each lung. Each enters its respective hilum, the most posterior of the structures forming the lung root, *i. e.,* two pulmonary veins, one pulmonary artery and one bronchus. They are in a plane anterior to the descending aorta, œsophagus, thoracic duct, etc.

Right Bronchus.—Usually it has a greater lumen than the left, and is practically a continuation of the trachea, owing to the obliquity of the left bronchus. About one-half an inch from the bifurcation the right bronchus gives off a large branch, which passes to and supplies the upper lobe of the right lung, terminating above in apical branches. It crosses above the right pulmonary artery, and is therefore eparterial.

All other branches of both bronchi are hyparterial.

The right bronchus, considerably diminished in size, passes to the hilum, and continues in the lung substance downward and backward, to terminate in the angle between the diaphragm and posterior parietes. It gives off four sets of branches (antero-lateral and postero-lateral), alternately placed. The first antero-lateral branch supplies the middle lobe; the rest, the lower lobe.

Left Bronchus.—The left bronchus passes more obliquely to the hilum of the left lung, and, entering the lung substance, terminates in the postero-inferior angle.

It gives off no branches until entering the lung, and then gives off four sets—four ventro-lateral and four dorso-lateral—alternately placed. Its first ventro-lateral branch differs from that of the right bronchus, because it gives off a large ascending branch, which supplies the upper part of the left lobe to its apex. It is therefore the homalogue of the eparterial branch of the right lung. The continuation of the first ventro-lateral branch supplies the rest of the left upper lobe.

The remaining ventro-lateral and dorso-lateral branches supply the lower lobe.

Referring to the X-ray picture of the bronchial tree, it is evident that more volume of sound passes down the right bronchus, because it has a greater lumen and is almost a continuation of the trachea.

The eparterial branch of the right bronchus arises just below the bifurcation, and, entering directly the right upper lobe, distributes its branches to the apex, passing through less lung substance than is the case with the ascending branch to the left apex. Therefore sound will be less modified on reaching the right apex than the left.

As breath sounds arise in the larynx, and are modified by the decrease in the lumen of the air-passages and surrounding tissue, we will expect to find in the right apex persistence of the tubular quality of the breath sounds. Such is the case, broncho-vesicular breathing being a normal condition on that side.

Increased vocal fremitus in the right upper lobe normally is also explained by the above anatomical facts.

Because the right bronchus is almost vertical, it is located in the right infrascapula region, and explains why frequently broncho-vesicular breathing is heard in that location.

Lungs. Right and Left.—Irregularly, pyramidal in shape. Each present an apex directed upward and placed deeply in the neck under the scaleni muscles; below, the base, deeply concave, resting on the dome of the diaphragm; an external surface markedly convex, fitting into the concavity of the ribs; an internal surface, concave to permit the lodgment of the heart and great vessels; an anterior border, sharply defined, overlapping the heart and great vessels; a posterior border, blunt, fitting into the concavity of the ribs between the posterior angles and the bodies of the first ten dorsal vertebræ.

Each lung is divided into two great lobes by an oblique fissure, which is practically constant. It begins posteriorly about three inches below the apex, and passes obliquely downward and forward on the external surface to the junction of the anterior border with the base. They reach inward almost to the hilum of each lung.

We see, therefore, that the upper primary lobe of each lung consists of the apex, anterior border and surface, while the lower primary lobe consists of the posterior border, posterior surface and base. The upper lobe of the right lung is usually subdivided into two unequal lobes by a secondary fissure running from the anterior border obliquely downward and outward to the great oblique fissure, thus cutting off a small triangular lobe from the anterior surface. The anterior border of the right lung passes nearly vertically downward till it reaches its base. The anterior border of the left lung leaves the vertical nearer its base than apex, and passes obliquely downward and outward to reach the sharp edge of the base, thus uncovering a small triangular area of the heart.

Surface Markings of Trachea, Bronchi and Lungs.—The trachea begins at the cricoid cartilage opposite the sixth cervical vertebra, and terminates deeply opposite the junction of the manubrium and gladiolus in front; fourth dorsal vertebra behind, or junction of the root of the spine of the scapula with its vertebral border when the scapulæ are normally placed.

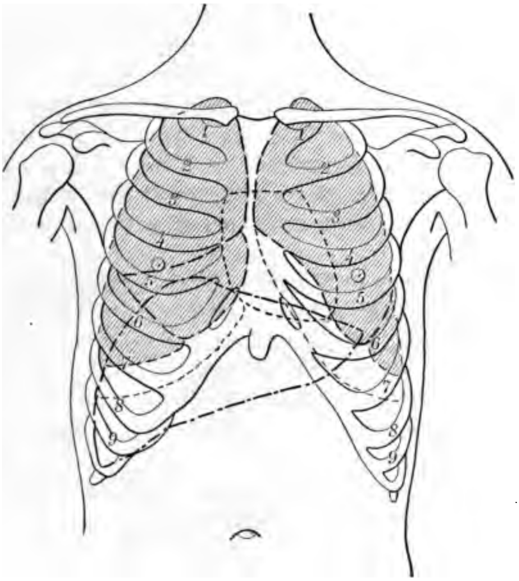
The bronchi enter the hilum of each lung about opposite the fifth dorsal vertebra.

Lungs.—The apex of each lung reaches into the supraclavicula fossa one to one and one-half inches above the clavicle, usually between the two heads of origin of the sterno-cleido mastoid.

ANTERIOR BORDERS. RIGHT LUNG.—Its anterior border approaches the median line opposite the second costo-sternal articulation, and continues downward in the median line to opposite the sixth costo-sternal articulation, where it meets the thin margin of the base. This margin leaves the median line, passes backward with a gentle convexity, pointing downward, and finally joins the posterior border at the tenth dorsal vertebra. In the mid-axillary line it usually crosses the eighth rib.

Anterior Border of Left Lung.—Reaches the median line opposite the second costo-sternal articulation; continues downward in the median line to opposite the fourth costo-sternal articulation. At this point it leaves the median line and passes downward and outward towards the apex of the heart, located in the fifth intercostal space, three and one-half inches to the left of the median line; and it continues to the sixth costal cartilage, where it reaches the thin basal margin. The latter then passes backward to the tenth dorsal vertebra, crossing the eighth rib in the mid-axillary line.

Fig. 3.

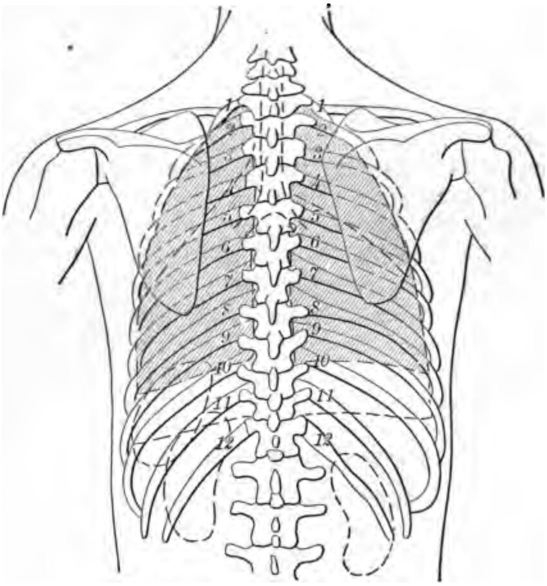


Relation of lungs, pleura, heart and liver to bony thorax.

Fissures. Right Oblique.—Begins at fourth dorsal vertebra, and passes downward and outward to the junction of the anterior border and base on the right se[illegible]al cartilage nearer its sternal articulation.

Left Oblique.—Begins at third dorsal vertebra and passes downward and forward to near the junction of the anterior margin and base, somewhere on sixth costal cartilage, external to the apex of heart. Therefore a smaller area of the left lower lobe presents anteriorly than the right lower lobe.

Fig. 4.



Relation of lungs, pleura, spleen and kidneys to bony thorax.

Lesser Fissure of Right Lung.—It leaves the median line opposite the fourth costo-sternal articulation, and passes downward and outward to meet the great oblique fissure in the mid-axillary line at about the sixth rib. (Figs. 3 and 4.)

PLEURA.

There are two distinct pleural sacs; one for each lung. They have no relation with each other except at the junction of the anterior margins of the lungs, when they slightly overlap.

Each pleural sac is divided into two portions, a visceral and parietal. The visceral pleura is in intimate relation with the lung wall, passing into the fissures. The parietal pleura is in intimate relation with the thoracic walls, pericardium, sides of great vessels, œsophagus, etc., as it leaves the under surface of the sternum to be reflected over these last structures in passing to the posterior parietes.

The parietal pleura becomes continuous with the visceral pleura at the root of the lungs and downward to the diaphragm, thus forming a double fold of pleura running from the root to the diaphragm, and called the lateral ligament of the lung. The parietal pleura leaves the base of the lungs and projects into the costo-phrenic sinus, reaching to the twelfth dorsal vertebra posteriorly.

Surface Markings of Pleura.

The surface markings of the pleura is that of the parietal pleura.

The right pleura begins at the apex of the right lung, one to one and one-half inches above clavicle. It reaches the median line opposite the second costo-sternal articulation, and continues vertically downward to the seventh costo-sternal articulation. It then crosses the seventh, eighth, ninth ribs, the tenth rib in the mid axillary line, and reaches the twelfth dorsal vertebra posteriorly.

The left pleura begins at the apex, one to one and one-half inches above the clavicle; approaches the median line opposite the second costo-sternal articulation, and continues in the median line to opposite the fourth costo-sternal articulation. It then deviates, skirting the border of the sternum about one-half to one inch from its left edge. It then has much the same course as the right. This deviation permits paracentesis pericardii without involvement of the pleura.

HEART.

The heart is a pyramidal-shaped organ of about the size of a man's fist. In the male it is five inches in the long diameter, three and one-half inches laterally and two and one-half inches antero-posteriorly.

It presents an apex, base, anterior and posterior surfaces, thin right border, due to the thin wall of the right ventricle, and blunt left border, due to the thick wall of the left ventricle.

It is obliquely placed, its base looking upward, somewhat backward and to the right; its apex downward, somewhat forward and to the left. It is anchored by its great vessels, which spring from the base, and rests by its posterior surface on the diaphragm.

The heart cavity is divided into four compartments: right and left ventricle, occupying the apical portion of the heart, and the right and left auricles, occupying the basilar portion.

The ventricles are asymmetrically placed, owing to the intraventricular septum passing from the base obliquely downward to the thin right border one-half to three-fourths of an inch from the apex.

The right ventricle consequentlv presents anteriorly, with its apex at the right thin border, nearer the sternum; while the left ventricle presents posteriorly, excepting its apex, which occupies the true heart apex, and therefore is farther removed from the sternum than the right ventricular apex.

The right ventricle presents an atrium and infundibulum, or conus arteriosus; the latter leading into the pulmonary artery. The left ventricle presents an atrium and aortic vestibule, the latter lying between the right mitral flap and ventricular septum. It is behind the infundibulum of the right ventricle.

Each auricle presents an atrium and appendix. The right auricle and appendix present anteriorly behind the sternum, and reaching to the right of its right border from the third to the sixth costal cartilages.

The left auricle presents posteriorly, its auricular appendix only presenting anteriorly behind the third rib about one and one-half inches to the left of the sternum.

The anterior auriculo-ventricular sulcis is placed obliquely behind the sternum, reaching from the left third costo-sternal articulation to the right sixth costo-sternal articulation. As the cardiac valves lie in the ventricular base, we see how their surface markings practically lie in this line.

The arch of the aorta begins at the base of the left ventricle, behind the infundibulum of the right ventricle, and beneath the inferior angle of the left third costo-sternal articulation; it passes upward and towards the right one and one-half to two inches. At the upper angle of the second right costo-sternal articulation it sweeps backward toward the left side of the fourth dorsal vertebra to become the descending aorta.

The pulmonary artery begins at the infundibulum of the right ventricle, behind the left border of the sternum, opposite the second interspace, and passes upwards, backward and to the left, where it bifurcates under the aortic arch into the right and left pulmonary branches.

Surface Markings of Heart.

Take a point on the lower border of the second costal cartilage, one inch from the left border of the sternum, and draw a line to the upper border of the third costal cartilage one-half inch to the right of the sternum. From this last point draw a line to the right seventh costo-sternal articulation, with a moderate convexity, to the right.

The apex is located in the fifth intercostal space, three and one-half inches to the left of the median line. Connect the left end of the base line with the apex, and the right seventh costo-sternal point with the apex, and we map out the entire area of the heart.

To differentiate this area, connect a point behind the left third costo-sternal articulation with a point on the right sixth costo-sternal articulation. This plots out the auriculo-ventricular sulcis. The heart area to the right of this line represents the position of the right auricle, the tip of its appendix being on this line opposite the third costal cartilage. The line running from the seventh right costo-sternal articulation to the apex of the heart represents the thin right border of the right ventricle. The left auricle is placed posteriorly, but its apex presents anteriorly at a point on the left convex line leading from the base to the apex, where it crosses the third costal cartilage about one and one-quarter inches from sternum. The continuation of this line represents the blunt left border of the left ventricle.

The Location of the Valves. PULMONARY VALVE.—It is beneath the upper angle of third left costo-sternal articulation. The valvular sound follows the course of the pulmonary artery to the pulmonary or left second interspace.

AORTIC VALVE.—Lower angle of third left costo-sternal articulation, just behind left edge of sternum. Its valvular sound follows the course of the aorta to the aortic or right second interspace.

MITRAL VALVE.—Behind the sternum, opposite fourth costo-sternal articulation. Its valvular sounds are carried to the apex of the left ventricle, or true apex of the heart.

TRICUSPID VALVE.—In median line of sternum, opposite fourth intercostal space. Its valvular sounds are carried to the apex of the right ventricle, or just to the left of the ensiform cartilage.

We observe that the valves are located nearly in the plane of the auriculo-ventricular sulcis, the auriculo-ventricular valves being located a little to the left of this surface marking, owing to the obliquity of heart; the apex pointing more anteriorly, the base more posteriorly.

Superficial and Deep Areas of Cardiac Dullness. — The superficial area of cardiac dullness is that portion of the heart uncovered by the lungs. It is triangular in area. The apex is above where the anterior borders of the two lungs separate in the median line opposite the fourth costo-sternal articulation. The right border of this triangle is the anterior border of the right lung, and therefore passes vertically downward in the median line. The left border is that of the left lung, and runs from the median line opposite the fourth costo-sternal articulation to the apex of the heart. There is no inferior boundary, as the cardiac dullness fuses with the liver dullness. The deep area of cardiac dullness is the rest of the heart outline.

PERICARDIUM.

A pyramidal-shaped serous sac, inclosing the heart, and the aorta and pulmonary artery for one and one-half inches of their course and the superior vena cava; but only partially enfolding the inferior vena cava.

Its apex is directed upward, where the pericardium continues as a tubular prolongation on the great vessels. Its base rests on the diaphragm, blending with central tendon and extending over the left muscular portion. It is composed of two portions, an outer fibrous coat and an inner serous coat, the latter lining the fibrous coat and reflected downward on the great vessels to enfold the heart.

The pericardium is attached to the sternum in front, and is in intimate relation with the parietal pleura, which is reflected upon it. It is covered by lung and pleura anteriorly, except for a small triangular area, where the left visceral pleura leaves the median line at the fourth costo-sternal articulation to be deflected to the left about two fingers' width from the left border of the sternum.

ABDOMEN.

The abdomen is divided into three zones—epigastric, mesogastric and hypogastric—by two horizontal planes. The upper one runs antero-posteriorly at the lower limits of the tenth ribs, while the inferior one passes between the anterior superior spines of the ilia. These three zones are subdivided by two vertical planes erected from the mid-Poupart points. The subdivisions are: right and left hypochondriac regions, which project behind the costal cartilages into the vault of the diaphragm, and the epigastric region, triangular in shape, within the subcostal angle. The right and left lumbar and umbilical region, and, lastly, the right and left iliac or inguinal regions and the hypogastric or pubic regions.

Liver.—The liver occupies the right hypochondriac region, part of the epigastric and the left hypochondriac as far as the mammary line.

Its upper limits fit into the vault of the diaphragm, *i. e.*, fourth intercostal space on the right and fifth on the left. Its sharp anterior border passes obliquely from the right hypochondriac region and below to the left hypochondriac region and above, crossing the right costal border between the ninth and tenth costal cartilage and the left costal border under the eighth costal cartilage.

The fundus of the gall bladder, when distended, presents under the right costal border where the lateral border of the right rectus abdominis crosses, or at the right ninth costal cartilage.

Stomach.—The stomach, when moderately distended, presents a cardiac or œsophageal entrance; a cavity divided into a fundus, projecting into the left dome of the diaphragm; a body reaching downward to the plane separating the epigastric from the mesogastric zone and an antrum pyloris, which is located to the right of the median line. It finally opens by the pyloric opening into the first part of the duodenum.

It occupies, therefore, the left hypochondriac region and a portion of the epigastric. The cardia and pyloris are deeply placed. The anterior surface rests against the anterior parieties. The stomach is in close relation with the liver superiorly, with the pancreas posteriorly; the left kidney and spleen to the left and posteriorly; the transverse mesocolon below.

The surface markings of the stomach are:

Cardia.—Left seventh costo-sternal articulation.

Fundus.—Reaches to fifth left intercostal space.

Greater curvature reaches to line connecting tips of ninth or tenth ribs.

Pylorus.—Just to right of median line, three and one-half to four and one-half inches below lower end of gladiolus.

When moderately distended, a triangular area of the stomach is palpable in front. Its right boundary is the free border of the liver, passing from under the right ninth costal cartilage to the left eighth costal cartilage. The left border is the left costal border from the eighth to the tenth. Its lower limit is the greater curvature, on a line crossing between the ninth or tenth ribs.

Spleen.—The spleen is in close relation to the stomach, being attached to its body by the gastro-splenic omentum. The spleen lies in the left hypochondriac region, but posteriorly placed. It extends between the ninth and eleventh ribs, its long axis running parallel to the left tenth rib. Its posterior limit reaches to within one and one-half to two inches of the vertebra, while its anterior limit extends to about the mid-axillary line.

The spleen lies in the hollow of the diaphragm, its upper third having the lung interposed between it and the parieties; its middle third separated from the parieties by the costo-phrenic sinus, a space below the lung, tween the diaphragm and thoracic wall. Its lower third is in contact with the left kidney and tail of pancreas. We thus see that it is practically impossible to palpate the spleen under normal circumstances.

Kidneys. RIGHT AND LEFT.—Retroperitoneal placed obliquely downward and outward on the posterior wall of the abdomen. The left is more superiorly placed, resting on the eleventh and twelfth ribs, and extending to within two and one-half to three inches from the iliac crest.

The right extends from the upper border of the twelfth rib to within about two inches from the crest of the ilium. The lateral borders of the two kidneys extends beyond the lateral limits of the erector spinæ muscles.

PART II.

THE RESPIRATORY SYSTEM.

CHAPTER II.

INSPECTION.

By inspection is understood the act of viewing the patient so as to learn all that may be observed by the sense of sight. Inspection is one of the most important methods of physical diagnosis, and all systematic examinations should begin with it; and while it is not final, it is valuable in determining certain essential facts that are further differentiated by the other methods of physical diagnosis.

The technique of inspection is as follows:

(1) The patient should be stripped of clothing as far as possible. In children and in the male adult all clothing should be removed from the chest except that which may be necessary to avoid chilling of the surface; in the female, delicacy may demand that the patient be thinly covered, but wherever it is required the examiner should not hesitate to demand that the surface be bare, if only a part at a time, so that correct observations may be made. Where for any reason it is impossible to bare the chest thoroughly, much of the observation that would ordinarily be made by inspection must be made by palpation.

(2) Inspection may be made with the patient either standing, sitting or in the recumbent posture, according to the circumstances of the case.

When the patient is able, the standing or sitting posture is preferable, inasmuch as it is easier for the observer, and the movements of the chest are not interfered with. The patient should assume a natural or customary posture. Usually as soon as inspection is commenced they try to assume a "correct" posture.

(3) It is necessary that the light fall equally on both sides, as the value of inspection is in the comparison of one portion of the chest with the corresponding portion of the opposite side. The observer generally stands directly opposite the patient, so as to have both sides within the range of vision. Where it is necessary to determine slight variations in the upper portion of the thorax, it can often be best done by the observer standing behind the patient and looking over the shoulder and downward along the clavicle and upper ribs. This will show the slightest degree of depression in these areas.

NORMAL CHEST.

The size of the normal chest varies within wide limits, and also with the age and sex of the individual. In determining whether or not its variations are within the normal limits, it is necessary to consider the general physique of the patient, as the size of the thorax should bear a definite relation to height and weight.

It is extremely difficult to describe a perfectly normal chest. The thorax should be well developed, although it is only one in four persons that have perfectly and symmetrically-developed chests. The neck should bear certain definite relations to the bony thorax, and, according to the general physique of the patient, the neck will be long or short and its relations will vary according to the development of the upper portions of the body.

The direction of the clavicles varies with the individual. In the broad, square-shouldered individual they lie almost in a horizontal plane, while in those with sloping shoulders the outer border tends to slope downwards and backwards to meet the tip of the shoulder blade. Above the clavicle there is, normally, a slight depression, called the supraclavicular depression. The depth of this depression varies in individual cases, and whether or not it is normal can only be judged by comparing it with the general contour of the chest. Increase in the depth of the depression above the clavicle occurs in those conditions which cause diminution of the size of the apex of the lung, interfering with its normal distension when the thorax is enlarged in respiration. These conditions may affect the entire lung, causing general contraction of the chest, as in general pulmonary fibrosis, etc., or may affect the apex only of one or both lungs. While a number of diseases may cause this local condition, the most important is pulmonary tuberculosis.

The sternum normally is carried forward and downward from its junction with the clavicle. At the junction of the upper and middle portion of the body the so-called angle of Louis is formed. This varies in individual cases, and is increased in certain diseases of the respiratory tract.

The ribs curve forward and meet the sternum so as to give a gradually increasing depth to the chest from above downwards. There is also a gradual increase in the angle with which they join the sternum from above downwards. At the epigastric notch the angle is well marked, and its acuteness varies with the general contour of the chest.

The intercostal spaces are of a definite width, according to the shape of the thorax. They should be slightly depressed, and in a well-developed thorax are visible only in the lower portions.

Posteriorly, the scapula lies flat upon the ribs, and the spine is, normally, slightly curved toward the right side.

The physiological departures from the perfect chest have been variously described as long and short, broad and narrow, deep and hollow. All these variations, though quite consistent with health, may give the student great difficulty in determining whether or not they are within the range of normal.

As the development of the chest varies greatly in different individuals, many chests are seemingly indicative of some intrathoracic diseases which depend upon non-development of the bony thorax and of the lungs during the maturing period of life.

How can it be determined if the shape, form and size of the chest is within the range of normal for the individual under observation?

This can be done only by taking into consideration not only the thorax, but the entire bony skeleton as well, and determining if the thorax harmonizes with it. The social status of the individual, the general physique, occupation, etc., must be fully considered. Faultv habits of posture may cause slight asymmetry of the bony thorax without in any way influencing the function of the contained organs. The observer must not form the inference that each departure from the ideal thorax is pathological, but must note the variations from the normal, and by the other methods of physical diagnosis determine if there is any change in the location, structure or function of the underlying thoracic organs.

Deviations from the normal chest have been classified as follows:

PATHOLOGICAL CHESTS.

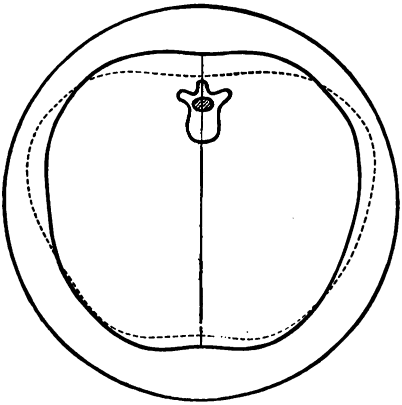
(1) **The Barrel-shaped, or Emphysematous Chest.**—In this form there is an increase in all of the diameters of the chest, especially marked in the antero-posterior. The chest assumes a type more nearly approaching that seen in the infant. (Fig. 5.) The position is one of full, forcible inspiration. The clavicle and sternum are carried upward and forward by the action of the auxiliary muscles of inspiration; the neck is shortened; the infraclavicular spaces may be deeper than normal, shallower, or may be even projecting. The scapulæ are

thrown upwards, outwards and forwards, and project from the rounded thorax. The normal antero-posterior curvature of the spine is increased.

Bilateral or uniform enlargement is most frequently caused by pulmonary emphysema (large lung); it occurs also during attacks of asthma (temporary emphysema), bilateral pleurisy with effusion, hydro-thorax and cancer (rare).

(2) **The Phthisical, Paralytic, Alar, or Pterygoid Chest.**—In this type there is an abnormal flattening of the sternum and ribs, so that the antero-posterior diameter of the chest is diminished and there is a slight increase in the lateral diameter. (Fig. 6.)

FIG. 5.



Bilateral enlargement of emphysema.

Outer line = a circle drawn to show how nearly the emphysematous approaches the circular shape.

Dotted line = natural adult chest.

Inner line = emphysematous chest.

Actual measurement in centimetres

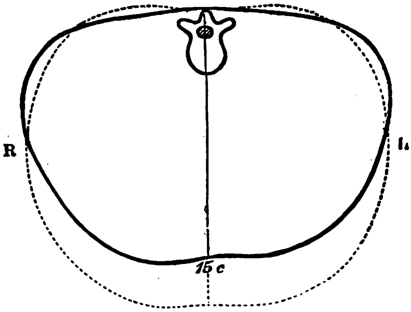
Circumference	**= natural, 89.0**	**emphysematous, 87.75**	
Transverse	**= " 29.6**	**" 27.25**	
Antero-posterior	**= " 22.25**	**" 25.4**	**(DR. GEE.)**

The depression of the ribs and of the sternum carries the sternal end of the clavicle downwards, lengthening the neck and causing the chin to project farther forward than normal over the anterior plane of the thorax, giving a characteristic appearance to the individual.

The angle of the plane of the clavicle with the scapulæ and sternum alters the depressions above the clavicles, increasing their depth. The intercostal spaces are deepened; the shoulder blades are depressed and stand out from the ribs, giving the winged appearance (alar, or pterygoid).

The movements on quiet breathing may be nearly normal in frequency and extent, but on exertion or forced breathing expansion is less than normal, and the movements become more rapid.

FIG. 6.

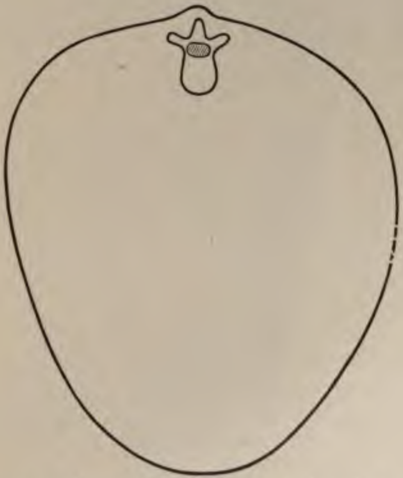


The flat or phthisical chest, short antero-posterior, long transverse diameter. (GEE.)

This type may be simulated in marked emaciation by diminution in the soft parts which normally give the rounded contour to the upper portion of the chest. Lack of muscular development, or changes in the muscles of respiration, such as occur from long recumbence in bed, cause slight depressions of the bony thorax, due to feeble inspiratory efforts. Symmetrical depressions of the thorax in the above conditions are easily differentiated from those dependent upon pathological intrathoracic conditions by noting that the normal contour of the bony thorax is preserved, although there may be a slight flattening of the chest.

The pathological conditions which tend to produce the phthisical type of chest are thickening of the intercostal pleura, which interferes with the elevation of the ribs, and widening of the intercostal spaces during inspiration; thickening of the pulmonary pleura preventing the normal expansion of the lung; changes in the lung which diminish its elasticity, as tubercular induration, fibrosis, etc., and the closure of the bronchi, which prevents the entrance of air into the lung.

FIG. 7.

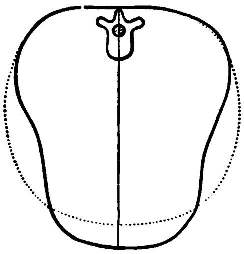


Transverse section of a rachitic chest at level of sixth thoracic vertebræ. Circumference, $32\frac{1}{8}$ inches; right half, $16\frac{1}{8}$ inches; expansion, 2 inches.

(3) The Rachitic Types.—In the simplest form there is a flattening laterally of the chest, especially in the lower segment; while in the upper portion the sternum is carried forward and the ribs are more or less straightened, so that the antero-posterior diameter of the chest is increased above, while the lateral diameter is diminished below. (Fig. 7.) From this simple type the diameters greatly increase up to the so-called "pigeon-breasted" type. (Fig. 8.) In this form the sternum is pushed forward; "the ribs are straightened out at their angle and at their junction with the cartilages," so that a section of the chest is nearly triangular and the whole contour resembles that of the breast of a pigeon.

Rachitic chests also frequently show nodular enlargements at the junction of the bony ribs with the cartilages, which have been named the rachitic rosary. (Fig. 9.) The depressions and peculiarities of the rachitic chest are due to intercurrent attacks of bronchitis, involving the smaller tubes and causing interference with the entrance of air into the alveoli, and the production of a partial vacuum during inspiration. The effect of atmospheric pressure and the action of the diaphragm is to depress the soft and yielding thorax.

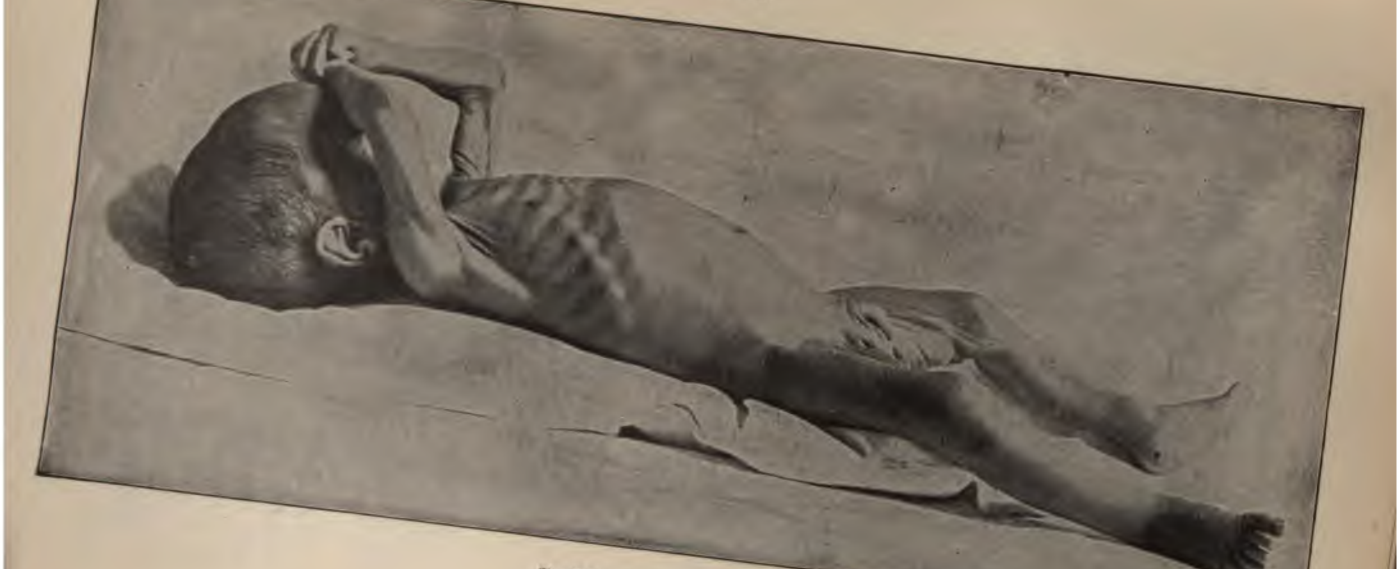
FIG. 8.



Circumference =42.75 centimetres.
Rickety chest. Dotted line indicates the shape of chest in an infant about the same age. (GEE.)

Frequently, near the junction of the fifth rib with the sternum, and running obliquely across the chest, there is a well-marked depression or groove, which has been named the Harrison furrow. The occurrence of

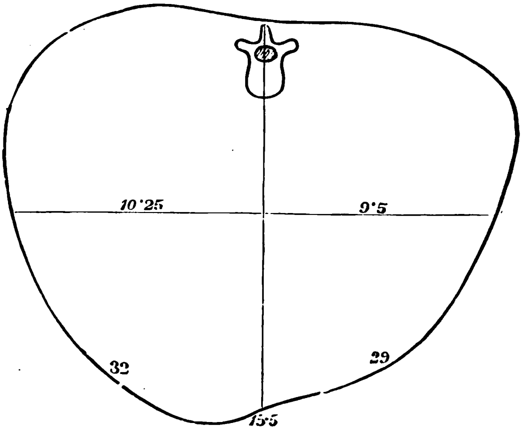
FIG. 9.



Rachitic rosary. (BARBOUR.)

Unilateral Diminution in Size.—In this condition the affected side is smaller in all dimensions. (Fig. 11.) The ribs are closer together; the intercostal spaces are narrowed, and may even be obliterated, or the ribs, in extreme cases, may overlap each other, especially in the lower portion. The ribs are more oblique than normally, giving that side of the chest a longer and narrower appearance. The mamma is nearer the median line; the top of the shoulder is lower than its fellow on the opposite side; the scapula is thrown nearer to the spinal column, and the spinal column is curved, the convexity looking towards the opposite side. The movements of the affected side are diminished, restricted, or may even be absent. The supraclavicular fossa is deeper than normal.

FIG. 11.



Unilateral retraction of chest, consequent upon cirrhosis of left lung, in a girl of fourteen years. The figures indicate antero-posterior and transverse diameters and semi-circumferences of right and left half of chest. (GEE.)

Unilateral diminution in size may be due to (1) primary arrest of development, as occurs in infantile hemiplegia, etc., when the asymmetry is not limited to the thorax alone, but generally extended over the entire lateral half of the body; and the affected side, although smaller than the opposite, preserves its normal physiological features.

Unilateral diminution in size also occurs when there is atrophy or loss of function of the muscles of normal inspiration. In this condition retraction of the side could only occur when the condition had lasted for a long time, and the unopposed traction or negative pressure of the lung had been sufficient to cause a gradual depression of the ribs.

(2) DISEASES OF THE PLEURÆ.—Marked retraction of the thorax occurs in diseases of the pleuræ, especially after absorption of the fluid in pleurisy with effusion, when changes occur in the lung or in the pleuræ which prevent the lung thoroughly filling the thorax, as pulmonary collapse and fibrosis and chronic thickening of the pleura, adhesions of the two surfaces of the pleura.

(3) CHANGES IN THE LUNGS.—These include changes in the lung which cause a diminution in its size, as chronic interstitial pneumonia (fibrosis, cirrhosis of the lung), tuberculosis, interference with the entrance of air into the lung through narrowing or occlusion of the lumen of the bronchi of entire lung.

LOCAL BULGINGS.

The asymmetry of the chest may be due to changes that affect a limited portion of the chest only.

Limited enlargements may be due to (1) changes that occur in the soft parts, as swellings or tumors, lipomas, malignant growths, etc.

(2) Diseases involving the bony thorax itself, as periostitis or exostosis, fractures of the bony thorax, or from localized deformities due to spinal curvature, distortion of the ribs, with enlargement of one side, the front of

which is compensated for by a corresponding increase of the opposite side behind.

(3) A bulging of a limited portion of the chest wall may be produced by a localized or encapsulated collection of fluid or air within the pleural cavity. The amount of deformity that such a collection may produce is directly dependent upon the conditions of the bony thorax, being most marked in early childhood, when the chest wall is soft and yielding, very slight effect or none being produced on the rigid chest wall of adult life.

(4) Bulging of the thorax over the præcordial space may occur as a result of cardiac enlargement, or of pericarditis with effusion. The amount of bulging will depend upon the condition of the chest wall, the same as was mentioned above. Dilatation of the large vessels may also cause local bulgings, which may be attended with visible pulsation. Local bulging of the bony thorax in aneurism does not occur until the pressure of the dilating vessel has been sufficient to cause a softening of the overlying bones.

(5) Enlargements of the lower zone of the thorax may be caused on the right side by enlargement of the liver, and on the left side by enlargement of the spleen.

(6) General enlargement of both sides of the lower portion of the thorax below the fifth rib may be produced by anything that increases the intra-abdominal pressure, as meteorism, ascites, abdominal tumors reaching the diaphragm, etc.

LOCAL DEPRESSIONS OR RETRACTIONS OF THE CHEST.

These may be produced (1) by diseased conditions of the soft parts, as local wasting in the muscles of a part. If there is paralysis of the muscle, so that its action upon the ribs is interfered with, the constant negative pressure that is exerted by the lung may be sufficient to cause a slight depression or retraction over the affected area. (2) Disease of the bony structures, as rachitis. (3) Changes in pleura, as thickening, adhesions. (4) It may be dependent upon a disease of pulmonary tissue, as tuberculosis, fibroid induration and other pathological conditions which render it less distensible.

It is necessary at this point to have a clear idea of the factors that produce localized depressions in diseases of the lung and pleura. In normal inspiration, when the thorax enlarges as a result of muscular action, the distensible lung readily fills the cavity. When, on the other hand, a change occurs within the thorax, affecting the pleura or the pulmonary tissue, either as a result of tubercular infiltration or fibroid induration, on inspiration the affected portion of the lung does not readily enlarge, and as a result there is a tendency to produce a vacuum at the point affected. In the early stages a compensatory dilatation of the surrounding alveoli may make up for this lack of distensibility of the lung, but, on account of the gradually-developing muscular weakness and constant action of the atmospheric pressure over the affected part, there is produced the characteristic depression or retraction of localized intrathoracic diseases.

In diseases of the pleura, characterized by adhesions and obliterations of the sac after the absorption of the fluid in pleurisy with effusion, with atelectasis of the lung, and in resection of the rib, these factors may cause a retraction of the entire side.

RESPIRATORY MOVEMENTS.

In addition to changes that occur in the form and size of the chest, the movements of respiration are to be noted. Difficulty may be experienced in getting patients to breathe properly. Under examination they are apt to breathe abnormally, and in certain nervous individuals, if they are conscious of observation, it is impossible to get a true idea of the respiratory movement. These individuals should be observed without attracting attention to your purpose.

Normally, the movements of the two sides are equal and simultaneous. With each inspiration the upper portion of the chest is well filled out; there is a movement upward and outward of the ribs; an increase in the vertical, transverse and antero-posterior diameter of the thorax; depressions of the intercostal spaces are increased, and there is a protrusion of the abdomen in the epigastrium, due to the descent of the diaphragm. With expiration the movements take place in the reverse order.

The movements of inspiration are active, depending upon the action of the muscles on the bony thorax, increasing the size of the thoracic cavity with the passive distension of the lung. In expiration the movements are caused by the elasticity of the overdistended lung, aided by the elastic tension of the entire thorax and the weight of the chest.

Three types of respiration are recognized as physiological:

(1) **The costal, or superior costal,** which is seen chiefly in women. In this type the movements of the upper portion of the chest are most marked; there is little expansion of the lower segment of the thorax, and the protrusion of the abdomen is slight. In this type the sternum as a whole is elevated. Elevation of the sternum occurs both in quiet and deep breathing in women, but in man only in deep breathing.

(2) **The inferior costal** in which the most marked movement occurs in the lower six ribs, and the diaphragmatic phenomena are more markedly noticed. This is the type of breathing normal for an adult male.

(3) **The diaphragmatic or abdominal breathing** which is chiefly present in children.

In determining whether the movements of respiration are normal, therefore, it is necessary to take into account the sex and age of the patient.

Costal breathing may be abnormally increased (1) when the movements of the lower segment of the thorax or diaphragm are interfered with by œdema or inflammation of the lower lobes of the lungs, preventing their expansion, in which case the exaggerated costal breathing is compensatory. (2) Pleurisy with effusion, involving both pleural sacs. (3) Diseases of the bony thorax, as Pott's disease, or painful affections which interfere with the movement of the ribs on both sides.

(4) Painful affections of the soft parts, although they rarely affect both sides. (5) Paralysis of diaphragm, due to bulbar paralysis, neuritis of the phrenic nerve in multiple neuritis, hysterical neuroses. When the diaphragm does not act there is absence of protrusion of the epigastric region, with inspiration, and it may be replaced by depression, which is especially noticeable in hysterical paralysis and diaphragmatic pleurisy. (6) Diseases below the diaphragm, with pain on movement of the abdominal organs, as peritonitis, general or local. (7) Increased abdominal pressure from ascites, tympanites or tumors, which mechanically prevent the descent of the diaphragm.

The *inferior costal and diaphragmatic breathing* may be increased by any condition that prevents or interferes with the normal costal breathing, as consolidation of the upper lobes of the lung from any cause, painful affections involving the pleuræ, intercostal nerves, or bony thorax. It is especially diagnostic if it replaces normal costal breathing in the female adult, except in old age. In proportion as there is a lack of expansion of the bony thorax and the movement of the ribs is interfered with, there is a corresponding action of the diaphragm and diaphragmatic, or abdominal breathing. In old age the bony thorax becomes more rigid, and increased abdominal breathing is present in the female as well as in the male.

The movements may be increased unilaterally whenever there is interference with the respiratory function of the opposite side. This condition is compensatory, the increased action of the unaffected side making up as far as possible for the absence of function of the other.

Increased Respiratory Movements.—The movements of respiration are increased in extent and number in conditions of dyspnœa, which may be due to (1) diseases of the lungs, which diminish their vital capacity, as bronchitis with exudation, consolidations of the lung from various causes, diseases of the pleuræ. (2) Cardiac diseases, in which there is imperfect æration of the blood from deficient circulation. (3) Diseases of the blood, in which its power to take up oxygen is diminished (anæmia). (4) Nervous diseases, in which there is no defect in oxygenation, but rather an increased irritability of the respiratory centers. (5) In those diseases in which there is an increase in the elimination of CO_2, as seen in fevers, etc.

Distinction is made between exaggerated breathing, or increased motion, in which the ordinary muscles of respiration only are involved, and labored breathing, where the extraordinary muscles of respiration are called into play.

Diminished respiratory movement may be bilateral, unilateral or local, and may occur as a result of (1) diseases of the muscles of respiration, (2) painful condition of soft spots, (3) diseases of the bony thorax, (4) diseased conditions of the pleuræ or (5) in diseased conditions of the lung, either general or local.

Bilateral diminution, or absence of motion, occurs when there is disease of the bony thorax, especially Pott's disease, preventing a normal movement of the ribs. Also in diseases involving the intercostal nerves and muscles. It is present, with enlargement of the thorax, when both pleural cavities contain air or fluid; when the elasticity of the lung is diminished, as in emphysema; after absorption of fluid in pleurisy or empyema; with thickening and adhesion of the pleuræ in diseases of the lungs which prevent or interfere with this expansion.

Whenever there is difficulty or interference with the entrance of air in the lung, through narrowing or occlusion of the upper air-passages or bronchi, as a result of forced inspiratory effort, there is a marked depression of the lower portion of the thorax, due to the partial vacuum that is produced. Not only is motion interfered with, but the number of respirations is diminished.

Unilateral and local diminution of motion varies in degree from slight lagging to entire absence of motion, and may be due to loss of muscular power; to painful conditions of the muscles (inflammation, neuralgia); of the bony thorax (periostitis, fracture of ribs, etc.); to diseased conditions of the pleuræ (acute pleurisy, painful and catchy breathing), or increased thickness and adhesions preventing movement of ribs and expansion of lung. These conditions are associated with unilateral and local retraction. When the pleura contains air and fluid, loss of motion is associated with increase in size.

LITTEN'S PHENOMENON.

In addition to the protrusion of the epigastrium, Litten has called attention to the effect that descent of the diaphragm has on the lower portion of the chest. If a person who is not too fat is so placed that lying on his back the light fall upon the chest from the direction of the feet only, the observer, standing at the side, notices a narrow shadow moving downward when a full, deep breath is taken. This shadow is due to expansion of lower ribs, caused by the slow separation, by the descent of the diaphragm, of the two surfaces of the pleura over the so-called complemental spaces and the gradual extension of the lower border of the lungs, with filling out of the intercostal spaces.

The phenomenon is modified or absent in all conditions that *(a)* change the normal relation of the two surfaces of the pleura in the complemental space, viz., effusion into the pleural cavity, pneumonia of the base of the lung, pulmonary emphysema; *(b)* or interfere with the descent of the diaphragm, as adhesions of the two surfaces of the pleura; or *(c)* prevents the lung from distending as fibroid induration and tubercular infiltration, especially of the apex.

Litten's phenomenon is often of value in diagnosing slight pleuritic effusions from enlargements of the liver and spleen, as subdiaphragmatic conditions do not interfere with the descent of the diaphragm, unless attended with pain or greatly increased intra-abdominal pressure.

ALTERATIONS IN FREQUENCY.

Rapidity of respiration varies normally with age. Under one year of age it is 44 per minute; one to five years, 26; five to twenty years, 20, and after twenty years of age the average rapidity is 18. Physiologically, it is influenced by posture, exercise, digestion and by mental influence. In diseases of the respiratory tract the rate is increased in proportion to the interference with the ærating function of the lungs. The rate in diseases of the lung is *never* decreased, except laryngeal or tracheal stenosis and asthma.

Diminution in the rate of respiration is otherwise dependent upon some influence acting on the nervous system, and especially on the respiratory center.

ALTERATION IN RHYTHM.

Normally, the movements of breathing are in perfect rhythm, each respiratory act being of equal length and depth and following each other without any appreciable pause. The movement of inspiration is slightly shorter than that of expiration, the ratio being as 5 to 6. As age advances this ratio becomes slightly greater, due to the impaired elasticity of the lung, so that in the aged a ratio of 5 to 8 is not unusual.

In asthma and emphysema the rhythm is disturbed in proportion to the expiratory difficulty, so that the movement of inspiration may be short, spasmodic or jerky, while expiration is slow, prolonged and incomplete.

When obstruction of the upper air-passages (intralaryngeal or intratracheal growths, false membranes, etc.) produce inspiratory dyspnœa, then the inspiratory movements are prolonged and the breathing is slow.

The rhythm is also disturbed when the respiratory movements cause pain (intercostal neuralgia, pleurodynia, pleurisy, etc.), and, as the pain is most intense at the time of greatest motion, the inspiration is short, shallow and catching and expiration slow and feeble.

A peculiar disturbance of rhythm has received the name of "Cheyne-Stokes" breathing. The movements are unequal and arhythmical, but follow a fixed cycle. One or two shallow respirations are followed by four or five that progressively increase in frequency, depth and noise until the acme is reached, and then gradually subside in inverse order. This is followed by a longer or shorter respiratory rest or "pause" (apnœa), which is followed by another attack of disturbed breathing.

This type of breathing occurs in severe types of cerebral, cardiac and renal diseases and narcotic poison, and is generally an unfavorable symptom.

CHAPTER III.

PALPATION.

By palpation we obtain information by the sense of touch, or tactile sense, and it is usually the second step in the examination of the respiratory and circulatory organs.

While it confirms all that has been learned by inspection, it is in many respects more definite and exact, and increases the evidence that has already been obtained by the eye, for it determines the presence or absence of certain vibratory phenomena and allows of differential diagnosis between conditions that produce similar changes in the shape of the thorax.

As inspection furnishes results of a general character, so also does palpation, and, on account of the ease with which it is performed, we often substitute it for inspection where that is impossible on account of the posture of the patient to view the chest.

The results obtained by palpation depend upon the sensitiveness of the observer's sense of touch *(tactis eruditus)*, and whether or not the chest is examined in a methodical and systematic manner.

Every part of the thorax should be examined, and those who claim that palpation is of minor importance are either deficient in the sense of touch or employ it in a haphazard manner without attention to details.

In performing palpation the surface should be bare, so the hand can be applied directly to the skin. It is not necessary to expose the thorax to view, as the hand can be slipped under a loose covering. At times it may be necessary, on account of a feeling of delicacy on the part of the patient, to have a thin covering, as gauze, between the surface and the hand, but even this interferes with the examination to a certain extent. The hands of the examiner should be warm, so as not to be unpleasant to the patient.

Attention should be paid to the position of the patient, and the examiner should always bear in mind that want of symmetry is a matter of greatest importance, so that the two sides should be compared region by region, as each portion of the chest gives its own evidence.

By palpation of the thorax, with reference to the respiratory organs, knowledge is gained of the following:

(1) SIZE, SHAPE AND SYMMETRY OF THE THORAX.

Palpation gives very little information on these points beyond what can be gained by inspection, and in most cases it is not as accurate in regard to symmetry. When inspection is impossible, on account of not being able to expose the chest, we gain a fairly accurate knowledge by palpation.

One important fact noted by palpation is the location of the different ribs. Remember that the second rib is the one below the clavicle, that can be taken between the fingers; it corresponds with the ridge on the sternum.

(2) RESPIRATORY MOVEMENTS.

As its object is to test if the movements of the two sides are simultaneous and equal, it is necessary that the hands be placed over corresponding regions of the chest for comparison.

As the time, extent and rhythm of the respiratory movements are to be examined, also whether the widening of the intercostal spaces occurs equally on both sides, the hands must be so placed as to note these points.

To palpate the anterior surface of the chest, the examiner stands behind the patient and places the hands over the chest so that the fingers are parallel with the ribs, resting in the intercostal spaces. By these means a better perception of motion and of time is obtained. To palpate the posterior surface, the position is reversed.

The antero-posterior movement of the chest is noted by placing one hand over the sternum in front, and the other posteriorly over the spine.

The movement of the clavicle and upper ribs is observed by placing the hand over the shoulder, the fingers resting on the anterior portion of the chest, and the thumb behind on either side of the spine.

The examiner should remember that the respiratory movements are influenced by age and sex, as already explained under "Inspection."

Slight inequality of motion between the two sides, as shown by a slight lagging of one side, when present in the slightest degree, is detected by the expert palpator, and is of diagnostic importance.

Under "Inspection" attention was called to Litten's phenomenon. The action of the diaphragm that produces it can be felt as well as seen, and gives us important evidence of any change in the movement of the diaphragm.

Palpation is almost as sensitive as the fluoroscope in detecting slight changes in diaphragmatic movement.

(3) VIBRATORY PHENOMENA.

Vocal fremitus, also called tactile and tussive fremitus, are the vibrations that are felt by the hand on the surface of the chest when the air contained in the lungs is thrown into vibrations *sufficiently strong* to be transmitted to the thorax and recognized by the sense of touch. The vibrations of ordinary respiration are too feeble to be appreciated by the hand, while they are recognized by the ear and termed normal vesicular murmurs, or breath sounds.

In order to obtain vibrations sufficiently powerful, the voice is used. The movements of the vocal cords set up corresponding vibrations in the air contained in the lungs, which are transmitted through the thorax and appreciated by the hand on the surface of the chest.

Vibrations set up by the voice are not of equal strength for all persons, varying according to the voice, sex and age of the individual. The wave length in man is from 8 to 12 feet; in women, 4 to 6 feet, while in the child the wave length is so short and the vibrations so rapid as to render vocal fremitus indistinct.

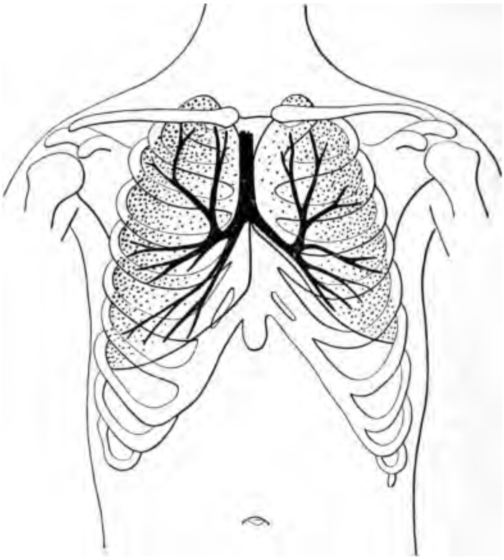
As the character of the vibrations produced by the voice modify the vocal fremitus that is obtained, it is necessary that the vocal vibrations be as equal and as uniform as possible. For this reason the patient is usually instructed to count 1, 2, 3 or use some phrase, as 99 or 44. Any monotone may be employed to set up the vibrations.

It must be borne in mind that each individual will have his own vocal fremitus, which will have a direct relation to the character of his voice. Voices that are low pitched and heavy, as in man, give a correspondingly strong vocal fremitus; while, on the other hand, the thin, weak or high-pitched voice, as in woman, being an octave above that in man, gives a correspondingly weak vocal fremitus, which in some cases may be so faint as not to be detected by touch.

The vibrations produced in the larynx do not reach all portions of the surface of the chest with equal intensity. We note that it is stronger over the right lung than over the left; that it is more marked over the right apex than over the left.

The reason for this difference in intensity is explained by the anatomical arrangement of the primary bronchi on the two sides and their relation to the trachea. The bronchus going to the right lung is more nearly a continuation of the trachea, has a greater lumen, and, in addition, leaves the trachea at a less acute angle, so that the vibrations that are set up in the larynx reach the entire right lung with greater force, on account of the larger column of air contained in the bronchi.

FIG. 12.



The situation of the bronchial tree emphasizes the fact that vocal fremitus is obtained more distinctly over the right than the left lung, greater in the upper than the lower thorax.

Vocal fremitus over the apices of the two lungs differ, being more intense on the right side.

This physiological increase of vocal fremitus on the right side often misleads the observer, as its intensity may suggest a pathological condition of the lung.

The cause of this marked increase of vocal fremitus over the apex of the right lung is due to the fact that the bronchus supplying the upper lobe is given off from the primary bronchus close to the trachea, and the vibrations conveyed to the upper lobe of the right lung are correspondingly stronger. Whether or not this increase is pathological can only be determined by considering other signs in connection with it.

The nearer the bronchi approach to the surface of the lung, and the larger their lumen, the more distinct will the vibrations be felt. For this reason the vibrations are stronger over the upper portion of the thorax, between the scapula behind, especially on the right side, and at the junction of the second intercostal spaces on the right side and the third on the left. (Fig. 12.)

The further the surface of the chest is removed from the larynx and trachea, the smaller the bronchi that supply the portion of the lung immediately underlying it, the greater the proportion of pulmonary tissue to the bronchi, the weaker will be the vibrations that are conveyed from the larynx to the surface of the chest, and consequently the vocal fremitus that is felt over the lower portion of the thorax and nearest the extremities of the lung is much weaker than over the large bronchi.

In addition to the influence of the pulmonary structure, the vocal fremitus is modified by the condition of the chest wall; and, according to the law that vibrations are lost in passing through media of different densities, and especially when passing from a media of a lesser to one of a greater density, vocal fremitus will be weak over those portions of the chest where a large amount of tissue intervenes between the hand and the pulmonary structure.

This is especially marked over the mammæ in women, over the scapula behind and is uniformly modified over the chest when the soft parts are greatly increased in thickness.

It is very necessary that the examiner should bear in mind the normal variations of vocal fremitus that occurs over different regions of the chest, and that he thoroughly familiarizes himself with their relative intensity.

In examining for vocal fremitus the whole hand is placed lightly upon the chest, so as to get a relative idea of the vibrations over the point that is examined. It will make a difference whether the fingers are placed in the intercostal spaces, as in testing for the respiratory movement, or on the ribs and bony thorax.

When the fingers are placed between the ribs, then the vocal fremitus that is felt is transmitted from the pulmonary tissue directly through the thin covering of the intercostal spaces, while if the hand is placed upon the ribs, or, as is usually done, across the ribs and intercostal spaces, then the vibrations that are felt are not those that are conveyed to the surface by the portion of the lung immediately under the hand, but are all the vibrations that have been brought to the surface and have been taken up by the bony thorax, which acts as a sounding board.

In order to examine for vocal fremitus in a limited area, it is best to place the tips of the fingers only upon the chest in the intercostal spaces while the patient is speaking. In this way slight local variations may be readily detected.

When the hand is placed over the shoulders, the thumb resting behind and the tips of the fingers beneath the clavicle, we obtain an idea of the relative intensity of the vocal fremitus that is produced in the entire upper portion of the thorax.

Vocal fremitus may be increased, diminished or absent.

Vocal Fremitus Increased.—As was seen above, under Normal Variations in Vocal Fremitus, the vibrations that are made in the larynx are modified by the conducting power of the lung, and are normally weakened as they pass from the larger to the small bronchi, and from these to the alveoli.

As the "spongy" condition of pulmonary tissue interferes with the transmission of vibration, so, on the other hand, any condition which renders the lung more homogeneous gives an increased power of conduction, and consequently increases vocal fremitus.

Increased vocal fremitus is obtained in all pulmonary consolidations, such as those of pneumonia, tubercular infiltration, infarction or fibroid thickening of the lung; and the amount of vocal fremitus is directly dependent upon the changed anatomy of the lung and its increased power of conduction.

As the vibrations that are brought to the surface are transmitted through the columns of air contained in the lung and the pulmonary structures, the tension of the lung tissue will modify the transmission of these vibrations.

Increase of tension increases the ease with which the vibrations are carried from one portion of the lung to the other, and also increases the rapidity of the vibrations themselves.

Increase of tension is present in pulmonary congestion, causing a slight increase in vocal fremitus.

In children, in addition to the high-pitched voice, with its attendant rapid vibrations, the tense pulmonary tissue causes the vibrations to become so rapid as to be indistinct.

Increased vocal fremitus is felt over cavities in the lung, as the vibrations from the larynx, trachea and bronchi are transmitted direct through the large air-containing spaces to the surface of the chest.

Diminished or Absent Vocal Fremitus. CHANGES DUE TO THE BRONCHI.—As the vibrations are conveyed chiefly through the columns of air in the bronchi to the surface of the lung, anything that diminishes the lumen of the tube would interfere with the transmission of the vibrations from the larynx in direct proportion to the diminution in size. If it is occluded, aerial vibrations are arrested, and there is an absence of vocal fremitus over the area supplied by the occluded bronchus.

Vocal fremitus may be diminished through narrowing of the lumen of the tube in bronchitis and asthma, and may be entirely interfered with in plugging of the bronchi, as occurs in fibrinous bronchitis, in pneumonia, or by the growth of tubercle nodules; and also by occlusion of the tube by pressure, as in aneurism.

CHANGES DUE TO PULMONARY TISSUE.—Just as tense pulmonary tissue by vibrating in unison with the air in the bronchial tubes increases the amount of vocal fremitus, so all conditions which render the lung tissue less tense interfere with the transmission of vibrations to the surface by smothering or absorbing the vibrations conveyed to it. This is especially noticeable with loss of elasticity and negative pressure in emphysema, while, on the other hand, it does not occur in compensatory emphysema, where the tension of the pulmonary tissue is increased, as in this condition the vocal fremitus may be normal, the increase of tension in the pulmonary tissue counterbalancing the increased sponginess from dilation of the alveoli.

INFLUENCE OF THE PLEURA.—Ordinarily the normal pleura, on account of its extreme thinness, has no influence on vocal fremitus. When, however, the pleura is thickened, or it contains effusion of any kind, it interferes markedly with the transmission of the vibrations from the lungs, according to the law that vibrations are lost when they pass from a rarer to a denser medium, and the amount lost, ranging from a slight diminution in intensity to total absence, will be in direct proportion to the amount of thickening or fluid that is contained within the pleural cavity.

The conditions of the pleura which interfere with the transmission of vibrations are: (1) Pleuritic thickening, which may be due either to primary pleurisy or be dependent upon tubercular infiltration: (2) Exudations upon the surface of the pleura, as occurs in acute pleurisy with plastic exudation: (3) Effusion into the pleura, whether serous, sero-fibrinous or purulent. The specific gravity of the fluid does not alter to an appreciable degree the transmission of vocal fremitus. (4)

When the pleural cavity contains air, as in pneumothorax, the vocal fremitus is interfered with, because the relaxed lung is a poor conductor of vibration.

INFLUENCE OF THE CHEST WALL.—Increase in the thickness of the soft parts will diminish vocal fremitus, according to the law that vibrations are lost in passing from a rare to a dense medium, but not to the same degree as a corresponding thickness of the pleura.

The reason for this is that when the pleura is thickened the vibrations are smothered or suppressed before they can be conveyed to the bony thorax, which acts as a sounding board and overcomes to a certain degree the inertia of soft (thickened) tissues of the chest wall.

This diminution will occur especially if the source of vocal fremitus, namely, the voice, is of such a nature as to give normally a feeble vocal fremitus.

Rhonchi and Palpable Râles.—In addition to the vibrations that are produced by the voice, the air passing through the liquid secretion in the bronchi is frequently thrown into vibrations sufficiently strong to be conveyed to the chest wall as fremitus. On account of being made in the bronchi, they have been named bronchial rhonchi, or palpable râles.

They occur chiefly with inspiration, and are most frequently felt in children; and are usually associated with noisy or asthmatic breathing, but the ordinary sibilant or sonorous râles of asthma are so high-pitched and the vibrations are so rapid that they are not detected by sense of touch.

Friction Fremitus.—Ordinarily the surface of the serous membranes glide over each other without friction. When, however, there is a marked roughening of the two surfaces, then their movement may be attended with vibrations sufficiently intense to be felt on the surface.

Friction fremitus is most commonly felt at the point of greatest motion of the serous membranes, normally in the axillary space over the fifth and sixth ribs.

Splashing or Succussion Fremitus.—When air and fluid are contained in the pleural cavity, and rarely in large cavities in the lung lying close under the pleura, on coughing or violent shaking of the chest the motion of the fluid contents may be sufficient to be felt on the surface.

Pain.—In addition to the vibratory phenomena, it is possible to determine by palpation whether or not pain is present.

Pain on palpation may be due to a sensitive condition of the chest wall itself, as is seen in the tender spots of Valleux, in inflammatory conditions of the muscular tissue and in diseased conditions of the bony thorax, as periostitis, etc.

An important variety of pain elicited by palpation is interpleural. In order to test for this it is necessary that the pressure be made so forcibly as to interfere with local expansion of the chest and bring the two surfaces of the pleura into close contact. The sign is valuable in tubercular involvement of the pleura over the apex. It is obtained by standing behind the patient, bringing the hands over the shoulder, as in testing for vocal fremitus, and making firm pressure with tips of the fingers in the infrascapular fossa.

Frequently it is possible to obtain this slight sign when no pleuritic râles can be detected by auscultation.

The test for pain, and especially interpleural pain, should be left till the last, as, if it is performed earlier in palpation, it is apt to disturb the rhythm and movement of the thorax, and so lead to false impressions.

Fluctuation. — By palpation we also determine the consistency and elasticity of tumors and enlargements of the chest that have been noted on inspection.

CHAPTER IV.

PERCUSSION.

PERCUSSION in physical diagnosis is the act of striking the body in order to elicit sound by setting up vibrations.

If the tissues that are percussed are solid, as the thigh, the sound produced will be dull, and is described as toneless, flat or dead. If, on the other hand, it contains air or gas, as the thorax or abdomen, it gives out a sound that has resonance or tone and the elements of sound—(1) quality or timbre, (2) intensity or volume, (3) pitch, (4) duration or length—will be present and easily recognized by the ear.

It is necessary to consider the elements of sound in detail, so as to appreciate their value in the percussion of the thorax.

The *quality* or timbre of a sound is "that characteristic by which the sound produced from some particular source, as from an instrument or voice, may be distinguished from sounds from other sources, instruments or voices."

It is physically dependent upon the form of the vibrations by which the sound is produced, and although the pitch, intensity and duration may alter, the quality still indicates the source of the sound.

The quality of the sound produced by percussion over lung tissue is called pulmonary or vesicular resonance.

The *intensity* or volume of sound depends upon the amplitude or extent of the vibrations. It varies directly as their square, and is further modified by the force of the blow.

The intensity is influenced in pulmonary percussion by the amount of the air-containing tissue that is set in motion. It is also modified by the surrounding tissues, whether or not they are easily thrown into vibration when struck, and so impart motion readily to the contained air. Therefore, in the percussion of the thorax the intensity of resonance is influenced by the condition of the chest walls, bony thorax and pulmonary tissue.

The influence of the force of the blow and the amount of air contained and the nature of the covering is readily illustrated in the drum. While the quality of the sound does not change, the intensity or volume is directly in proportion to the force of the blow upon the drum head.

The *pitch* depends upon the rapidity and length of the vibrations. The shorter and more rapid the vibrations, the higher the pitch. This is well illustrated in the fiddle string or drum head. The pitch becomes higher when the tension of the vibrating medium is increased, and is lowered by relaxing it.

The pitch is the most difficult part of a sound to appreciate, because the ear does not note slight variations in pitch as readily as changes in quality, intensity or duration, and it is only by training the ear that the finer changes are recognized.

Beginners are confused by the fact that high pitch is most frequently associated with dullness, and therefore it seems impossible to distinguish between a high-pitched note which is dull or characterless, as over-consolidated lung, and a resonant, clear note that is also high pitched, as occurs over distended lung tissue with increased tension.

Within certain limits, pitch varies in the lung according to the tension of the tissues, but, as this is slight, it can only be considered in connection with the other elements of sound.

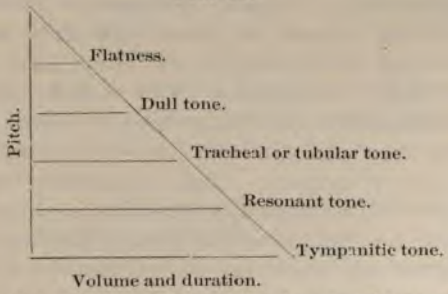
The pitch of the percussion note over normal lung tissue is low, varying within narrow limits in different individuals.

As pitch depends not only upon the rapidity and length of the vibrations of the air in the pulmonary tissue, but also upon the facility with which the vibrations pass through the tissues, the different notes we obtain in percussion of the thorax are modified by the soft parts of the chest, the bony thorax and the pathologic conditions in the pleura and pulmonary tissue.

From a diagnostic standpoint, variation in pitch is the most important of all changes that occur in the percussion sound, as it shows the physical condition of the part percussed.

The *duration* of sound depends upon the length of the waves and their persistence, and varies directly with the pitch and intensity.

FIG. 13.



Diagrammatic sketch of the relations of the character of tone. The perpendicular line represents the pitch. The transverse line the volume and duration.

The elements of sound have a definite relationship to each other. (Fig. 13.) Sounds that have the highest pitch have the least intensity and minimum duration and resonance. Such a sound is described as flat or airless.

As the pitch becomes lower the intensity increases, duration lengthens, quality of resonance becomes more marked and the sound is described as dull, resonant or tympanitic, according to the pitch.

Normal pulmonary resonance has a quality characteristic enough to be easily recognized, although impossible to describe, low pitch, great intensity and duration.

It is this combination of the elements of sound that is described by the "clearness" of the note.

As these elements vary, the various pathological types are produced.

METHODS OF PERCUSSION.

In order to elicit sounds from the air-containing spaces of the lungs by percussion, vibrations are set up in the chest in a number of different ways:

(1) Immediate, or Direct Percussion.—This was first used by Auenbrugger, in 1761. In this method the blow is struck directly upon the chest wall, generally over the bony structures. The sound produced is chiefly that made by striking the bony thorax, and its value is to show the ease with which the thorax itself is thrown into vibration. Light immediate percussion over the clavicle is especially sensitive in showing slight differences in resonance at the apex of the lung in beginning tuberculosis when no change can be detected by mediate percussion.

(2) Mediate, or Indirect Percussion.—In this method the blow is struck not directly upon the thorax, but upon some interposed medium, which is called the pleximeter. In this country a finger of the left hand, either the index or the second, is generally used as the pleximeter, but some examiners use in its place pleximeters made of various substances, as ivory, wood or other material that has a certain amount of elasticity.

It is important that the student become familiar with a single method. If he is constantly substituting for the finger the ivory or wood pleximeter, he does not become proficient in any one method, and is confused by the sounds elicited. Ordinarily one of the hardest things for the student to ignore in percussion is the sound produced by striking the pleximeter, which, in instruments made of ivory, etc., is especially marked, and is one of the chief objections to their use.

The finger, on the contrary, has a structure homogenous with that of the thorax, and does not add a new quality to the percussion note. After a certain amount of practice, this sound is ignored, and only that which is elicited from the deeper portion of the thorax noted. As a pleximeter, the fingers give a wider range in size and shape, and readily adapt themselves to the surface of the thorax.

It is a matter of choice which finger of the left hand is selected for this purpose, as all have been advised, but whichever is selected, that one should always be used. The other fingers and the hand should be raised from the chest so as not to dampen the vibrations. The finger used should be applied firmly enough to hold the soft parts in place and to leave no space between the finger and the soft parts, as this will give an impure or "cracked-pot" sound.

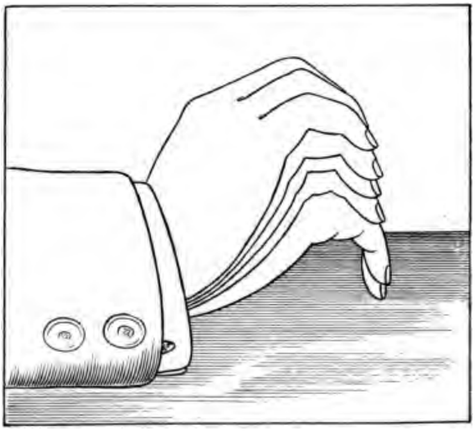
In addition to the above advantages, the finger also allows the examiner to note the resistance of the tissue percussed and any lack of vibration, so that in percussing the chest one frequently feels more than he hears.

Accurate comparative percussion requires that the finger be applied to corresponding spots on the two sides. If percussion on one side is over the rib or interspace, it should be made over the same tissues on the opposite side, and the same amount of pressure should be used.

To obtain the purest pulmonary resonance, the finger should not be placed upon the bony thorax, but in the interspace. The reason for this will be considered later.

That with which the blow is struck is called the plessor. For this purpose may be used the finger or fingers of the right hand, or a small hammer of ivory, wood or some other elastic substance. Many hammers have been advised, and the virtues of each one has been extolled by the inventor, but all are inferior to the finger, although proficiency in finger percussion is harder to attain, and demands no little practice. Ordinarily the failure to appreciate the value of percussion as a means of diagnosis is due to imperfect technique. The fingers are bent in a crooked and uneasy position; the whole arm is then moved, and the blow is struck from the elbow. This blow is necessarily a heavy one, and, on account of the infrequency with which it is struck and the length of time that the percussing finger rests upon the pleximeter, suitable vibrations are not set up in the chest wall.

FIG. 14.

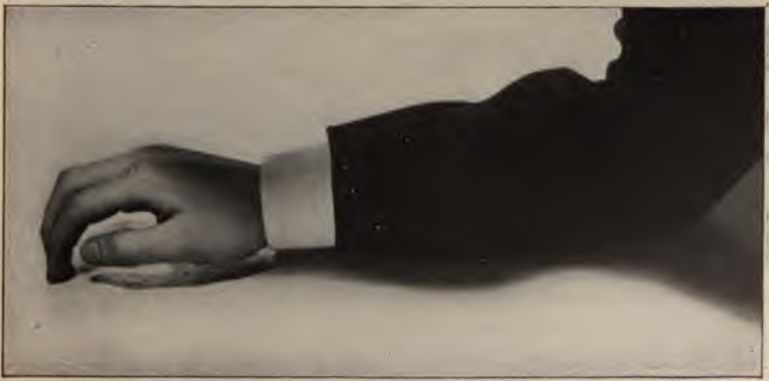


Showing position of fingers in percussion.

To get the clearest sounds, it is necessary that the fingers be bent from the second joint, so that the tips of the fingers are even, and they must strike squarely and quite vertically upon the pleximeter. The blow should be struck entirely from the wrist, and should be a short, sharp tap, the percussing finger or fingers resting but a short time upon the finger struck. (Fig. 14.) Light percussion can be performed by a motion of the finger only. Care should be taken by the beginner that the nail of the hammer finger does not strike directly upon the underlying finger. The blow should be struck with the rounded end of the finger, just as it curves to meet the palmar surface.

A good way of obtaining the wrist motion is by placing the entire arm flat upon the leaf of a table and striking it from the wrist from forty to eighty times a minute. (Figs. 15 and 16.) The resonant table leaf readily shows by the sound any variation in the force or frequency of the blow. It is well also for the student to stand close to a wall, facing it, and, applying the arm to the wall, perform the same motions, so as to accustom himself to percussing in different positions.

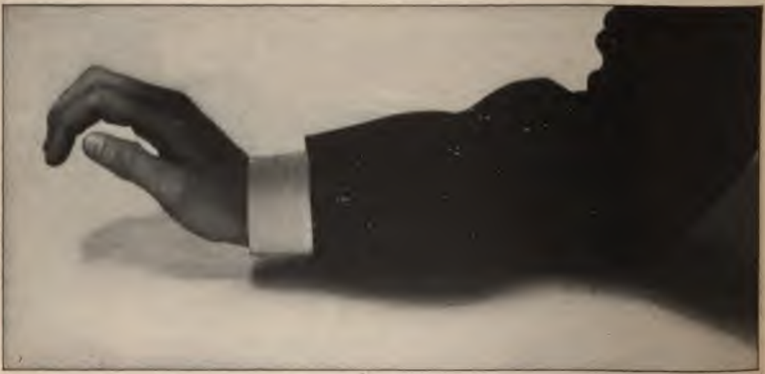
FIG. 15.



The rapidity with which the blow is struck has a marked influence upon the amount of sound produced. In certain cases single blows, with long intervals between, are used for the purpose of determining the ease with which the thorax is thrown into vibration. When they are struck with medium rapidity (but not less than forty to the minute), the vibrations reach the deeper portion of the lung with equal strength and a clear note is obtained. When the blows are struck too rapidly, there is an accumulation, so to speak, of vibrations, and there occurs an interference of the sound waves.

The force of the blow should be the same over corresponding portions of the two sides.

FIG. 16.



A correct position for the patient is almost as important as the technique of the operator. The surface should be bare, but if this is undesirable, then the covering should be as thin and soft as possible. Males do not object to being stripped to the waist for examination, but in the female, and especially the young, it is best to have the surface protected by a soft, thin dress, sack or shawl.

Whether standing, sitting or lying, the patient should assume a perfectly easy and natural position, with the muscles relaxed and the positions of the two sides symmetrical. While the standing posture is the most convenient for the examiner, it is best to examine patients who are weak in either the sitting or lying position, as the pressure of the finger on the chest is apt to cause them to sway, or, to prevent this, they brace themselves, and so cause uneven tension of the muscles of the two sides. In examining patients in bed, care should be taken that the body is straight and the shoulders not unevenly placed.

It should be remembered that when the patient is in bed and surrounded by pillows, the character of the percussion sound will be somewhat changed, being slightly muffled; also that the percussion outline of the organs is modified by their "passive mobility."

When percussing the front of the thorax, the patient's arms should hang easily at the side, and the head should be held straight in the median line. The patient, in order to avoid breathing in the examiner's face, frequently turns the face sharply to one side, rendering percussion above the clavicle difficult, and the resulting tension of the muscles changes the sound of the two sides. To percuss the axillary space, the arm should be moved slightly backward, or raised at a right angle to the body, with the hand resting on the head, so as to relieve the tension of the muscles.

When percussing the back, as the heavy muscles and scapula interfere with the pulmonary tissue underneath, the shoulder blades are carried forward toward the axillary space by folding the arms across the chest, with the tips of the fingers resting on opposite shoulders. Or, when sitting, the arms may hang loosely between the knees, allowing the shoulders to drop forward as much as possible. These positions uncover a large portion of the thorax, and the line of the scapula corresponds to the lower border of the upper lobe and interlobular septum.

CONDITIONS MODIFYING PERCUSSION SOUND.

As the object of percussion is to elicit sound from the deeper portion of the thoracic cavity, it is necessary to consider the influences that the different structures have upon the vibrations that are set up.

(1) **Influence of the Soft Parts.**—The ease with which the soft parts are thrown into vibration depends largely upon their structure. Muscular tissue, on account of its tension, is easily influenced, so that, with the bony thorax chiefly covered with muscle, vibrations are readily conveyed to the deeper portions of the chest. On that part of the thorax where the muscle tissue is heavy, blows that would be sufficient to set up vibrations in the air-containing spaces below the thinly-covered portion are absorbed by the heavy muscle, and no sound is heard.

Adipose tissue, on account of its lack of elasticity, is thrown into vibration with difficulty, while œdematous tissue is the most difficult of all to influence by ordinary percussion. The effect that the soft parts have upon vibration is clearly shown in Fig. 17.

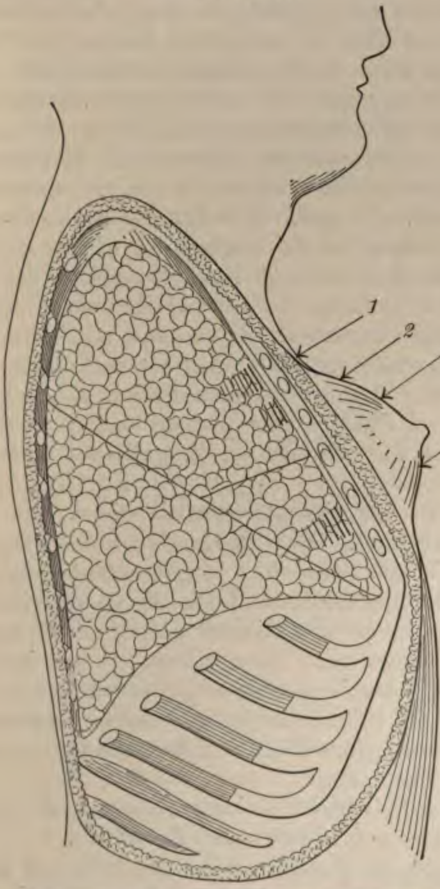
In order to overcome this interference of the soft parts with the transmission of vibration, it is necessary that the percussion be more forcible, as shown in Fig. 17.

It is thus easily perceived that percussion of the same strength will not elicit the same sound over all portions of the chest, irrespective of the condition of the lung itself. Where muscle or adipose tissue is thinnest a fairly good pulmonary resonance may be obtained with light percussion, while, on the other hand, where the muscle or fat is thick, as over the mammæ and the heavy muscles of the back, very forcible percussion is necessary, and the sound obtained is the dull, high-pitched note of the solid structures with little pulmonary resonance.

The student must be on his guard to differentiate between dull, high-pitched notes that are obtained by percussion over the thick soft parts from the dullness that is due to intrathoracic changes.

(2) **The Influence of the Bony Thorax.**—Bony tissue,

FIG. 17.



1, 2 and 3 represent the difference of result of percussion notes of equal strength, but with different body thicknesses. 3 and 4 show how by increasing the force of the blow lung resonance is obtained.

when thinly covered, gives a peculiar note of its own when struck. The bony thorax is easily thrown into vibration, and if the blow be struck upon a rib the vibrations that are set up are not only transmitted to the viscera underneath, but are conveyed along the ribs to the sternum and other bony portions of the thorax.

The readiness with which the bony thorax takes up vibrations must also be considered in the force of the blow. If the blow is very forcible, the whole of one side of the thorax may be thrown into vibration, and then, instead of obtaining sound from the portion immediately underneath the part struck, vibrations are obtained from the whole of the thorax, the sternum acting as a sounding board. Forcible percussion over the clavicle, on account of the conveyance of the vibrations to the sternum and through it to the general thorax, is used merely to indicate the condition of one side of the chest as compared with the other, and is of little value as indicating the condition of the tissue immediately underneath it. A marked consolidation of one lung, or a collection of fluid in the pleural cavity, makes the percussion of the clavicle on the affected side much duller.

Percussion over the sternum is resonant, with a peculiar bony note. The sternum acts as a sounding board, and is influenced to a slight extent only by the normal underlying tissue. When the normal resonance of the sternum is diminished and a dull sound is obtained, it shows that these vibrations are interfered with by intrathoracic conditions, that replace the anterior edges of the lung, solid growths in the mediastinum, by fluid accumulations in pericardium, or by aneurysmal dilatation of the aorta.

The ease with which the bony thorax is thrown into vibration varies at different periods of life.

In the child, on account of the flexibility of the ribs, vibrations are easily conveyed to the underlying lung, and a clear, well-defined pulmonary resonance is obtained, scarcely modified by the sound given off by the bony thorax. Percussion in children should, therefore, be light, otherwise diffuse vibrations are set up and resonance is obtained from a large extent of lung tissue.

As the bones of the thorax become more rigid, especially in old age, the bony sound becomes more marked, and there is a raising of pitch, so that it is described as a boardy or wooden note.

(3) **Influence of the Pleura.**—The normal pleura has practically no effect on the vibrations produced by percussion, but pathological changes which cause thickening influence them in a marked degree.

On account of the close relation that the pleura bears to the ribs, a very slight thickening is sufficient to interfere with the vibrations that are set up by percussion of the overlying structures. This is detected not only in the sound elicited, but in a peculiar "hard feeling" imparted to the fingers. The ordinary springy condition is changed to one of increased resistance, and varies in degree according to the characters of the changes in the pleura.

When the thickening is caused by new tissue homogeneous with the pleura, then its influence is slight. If, however, a thick layer of plastic exudation covers the surface, then the dullness will be in direct proportion to its thickness.

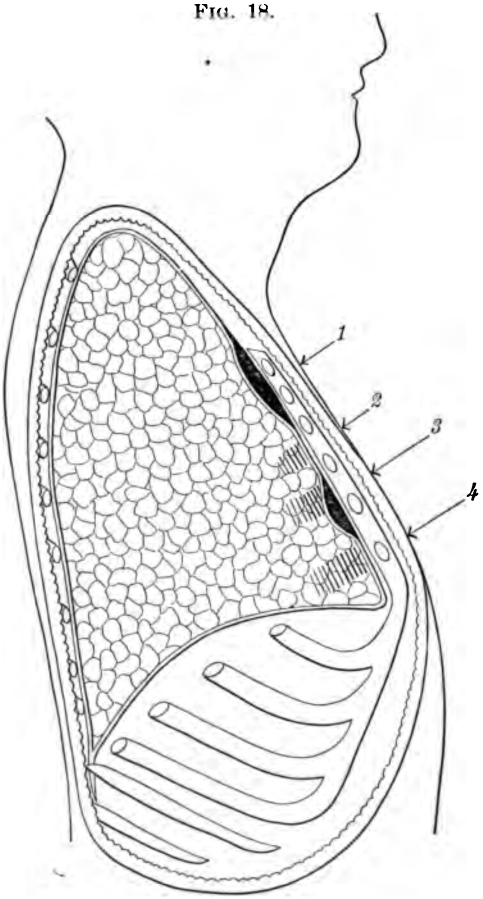
A collection of fluid within the sac renders the note flat, the feeling being similar to that noticed when the thigh is struck.

In order to determine whether or not the variation in the resonance is due to pleuritic thickening or to changes in the pulmonary tissue, the effect of percussion of different degrees of force must be used.

Light percussion will be markedly influenced by slight pleuritic thickening, while in very forcible percussion the vibrations are so strong as not to be interfered with to any appreciable extent. This is shown in Figs. 18 and 19.

(4) **Influence of the Pulmonary Tissue.**—The resonance that is obtained from the thorax is due to the lung being an air-containing organ, and the elements of sound depend largely upon the condition of the tissues of the lung itself and the amount of air contained.

FIG. 18.



1 and 2 show effect of weak percussion over pleuræ. 3 and 4 show effect of strong percussion over pleuræ.

The walls of the alveoli, on account of their marked distension and the tension, are easily thrown into vibration, giving a sound with a characteristic quality, low pitched, of great intensity and duration—the normal pulmonary sound or resonance.

(a) CHANGE IN TENSION.—*Increase in tension* of the pulmonary tissue causes an exaggeration of the normal resonance. The two conditions in which this occurs are in acute compensatory emphysema of the lung before the duration of the distension has allowed of permanent dilatation, and in children where there is marked elasticity of the alveoli walls and relative overdistension of the lung.

The slight change in the tension of the lung that occurs at the end of full inspiration and expiration causes a slight variation in the resonance. It is most marked at the borders of the lung.

Decrease in the tension of the pulmonary tissue gives a tympanitic quality to the sound. The change in tension may involve both lungs, and be due to pathological changes in the pulmonary tissue, decreasing its elasticity, as in emphysema.

The tension of the lung is also lowered by marked upward displacement of the diaphragm, as it occurs in increased intra-abdominal pressure from ascites, tumors, etc. The tension of the lung is also relaxed by any accumulation of fluid in the pleural sacs. In old age not only is the pulmonary elasticity diminished, with a lowering of tension, but the rigidity of the thoracic wall causes the percussion note to have a peculiar tympanitic quality.

The changes in tension may be local, as occurs over the affected area during the first and third stages of croupous pneumonia and in the unaffected portions of the lung on the same side during the second stage, due to enlargement of affected portion. This is most marked when the entire lower lobe is involved. When the pleural cavity is sufficiently filled with fluid to allow of relaxation of the lung, the same condition occurs. The peculiar tympanitic resonance over the relaxed portion of the lung in the second stage of pneumonia, and in pleurisy with effusion, has been named Skoda's resonance.

One of the earliest signs of tubercular infiltration of a portion of the lung, especially when it affects the apex, is the slight tympanitic quality of the percussion note.

The influence of diminution in the pulmonary tension upon the percussion note of the thorax demands further consideration.

In the normal chest, when the blow is struck upon the bony thorax, the negative pressure or suction action of the lungs causes a movement inward of the ribs, while at the same time it prevents a corresponding free outward rebound. This has the effect of dampening the vibrations of the bony thorax. The sound obtained is that of the pulmonary tissue, modified to but a slight extent by the peculiar sound of the bony thorax.

With diminished elasticity of the lung and decrease in the negative pressure, when the bony thorax is percussed its movements are not influenced to the same degree by the lung, and the percussion note contains a larger proportion of the bony quality and pitch; while at the same time the pitch of the pulmonary resonance is lower, and the sound obtained is described as slightly high pitched, wooden or boardy.

In percussing the thorax in diseases in which a lowering of the tension occurs, the examiner must exclude the bony quality of the sound and heightening of the pitch that occurs.

(b) Change in Amount of Pulmonary Tissue.—*Increase in the amount* of pulmonary tissue in any portion of the lung, whether or not it diminishes the air-containing space, produces a change in resonance. This change is described as dullness, and is characterized by heightening of the pitch and a peculiar hardening of the quality, with diminished intensity and

duration. If the size of the air-containing spaces be decreased at the same time that the solid structures of the lung are increased, the diminution of resonance would be in direct proportion to the changes. When the tension of the tissue is higher than normal, the pitch will be markedly raised; while, on the other hand, if there is marked relaxation, the dullness will be attended with a lowering of the pitch and will have a tympanitic quality.

Increase in the tissue of the lung relative to the air-containing spaces occurs with fibrosis, where the normal structures are increased either as a result of germ infection, especially by the tubercle bacilli, or of mechanical irritation, as in the conditions classified under pneumoconiosis.

(c) CHANGES IN THE AIR-CONTAINING SPACES.—*Increase in the size* of the air-containing spaces and the amount of air contained in the lung causes a marked augmentation of the resonance produced by percussion, and the quality of the sound will depend upon the physical condition of the pulmonary tissue. With the enlargement of the air spaces, if the tension of the pulmonary tissue is increased, as occurs in compensatory emphysema and in acute asthma, the pitch is slightly raised and the sound is abnormally clear. When, on the other hand, the pulmonary tissue is relaxed, as in large lung and in the senile form of emphysema, the pitch is lowered and of a tympanitic character, giving the typical emphysematous or "band-box" percussion note.

When destruction of a portion of the lung tissue occurs, forming large air-containing spaces or cavities, the percussion sound will depend upon their situation, size, form, condition of the walls and the surrounding tissue, and whether or not it is open or closed and the amount of fluid contained.

Cavities lying close to the surface, and especially at the apex, and opening more or less directly into a medium-sized bronchus, give the most marked sound, and those the size of a walnut can be detected by percussion. When situated deeper in the lung, and surrounded by fairly normal lung tissue, only a faint tympanitic quality may be noted. When covered by thickened lung tissue, or by thickened pleura, the sound becomes dull and at the same time slightly tympanitic. This has been variously described as boardy tympanitic, dull tympanitic, etc. The influence of the overlying tissue may be so great that no cavity sound can be obtained. (Fig. 20.)

When the walls of the cavity are rigid, the pitch of the sound changes. In those with lax walls the sound is described as *cavernous*. The quality is tympanitic; the pitch is low, with intensity and duration proportionate. In those with tense, rigid walls it is described as *amphoric*, with a more marked resonance and higher pitch. These variations in sound are comparative; the cavernous merges into the amphoric, and numerous subdivisions have been made, as caverno-amphoric, etc.

When the bronchus leading to a cavity is closed, the tympanitic sound is less marked, and the sound is duller and high pitched.

Certain peculiar changes have been detected over cavities:

WINTRICH'S CHANGE OF SOUND, OR FORCIBLE PITCH.—In cavities opening into a large bronchus, and situated near the surface of the lung, a change of pitch is noted when percussion is made with the mouth open or shut. When the mouth is wide open, it is high pitched, and more tympanitic than when the mouth is closed. Frequently cavities that give no distinguishing feature when the mouth is closed are easily detected when the mouth is open.

GERHARDT'S CHANGE OF SOUND.—At times the pitch and intensity of the tympanitic sound over a cavity changes with the posture of the patient. Various explanations have been given of this phenomenon, but it is probably due to alterations in size and form of the cavity, its relation to the chest wall and the connecting bronchus. The sign is not often detected, but when present is diagnostic of a cavity.

FRIEDRICH'S RESPIRATORY CHANGE OF SOUND.—This is noted when percussion is made during forcible inspiration and expiration, and is dependent upon the changes that occur in the size of the cavity and the tension of its walls.

THE CRACKED-POT SOUND.—This is a peculiar "clinking" sound heard when forcible percussion is made over a cavity connecting directly with bronchus during expiration with the mouth open, and is most easily obtained when the cavity is situated in the upper portion of the lung and the chest walls are flexible. This sound can be imitated by loosely clasping the hands with palmar surfaces slightly touching and forcibly striking the back of one hand on the knee, thus forcing the air through the narrow chink formed. This sound, while most frequently found in cavity formation, may be produced by forcible percussion in the normal lung when the thorax is very elastic, as in children.

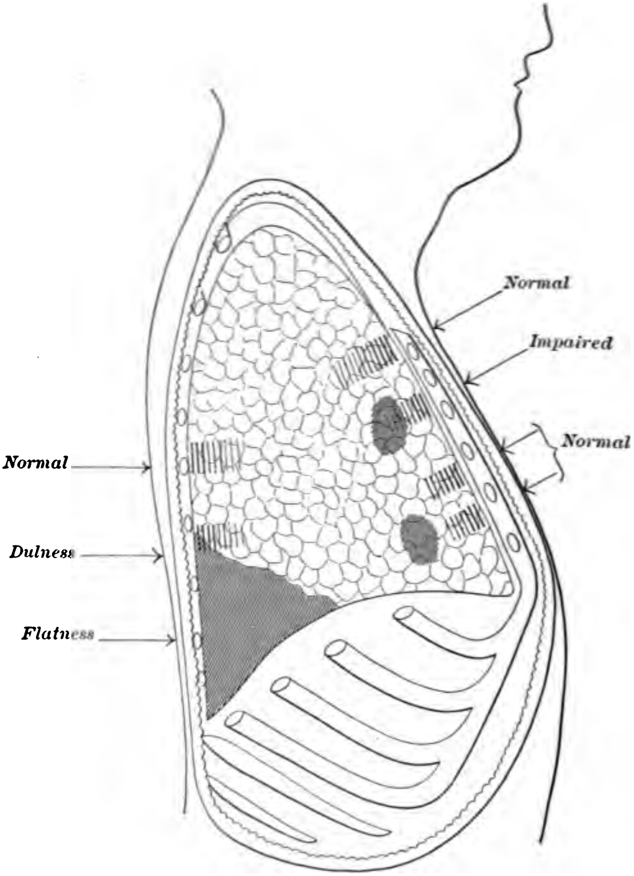
Decrease in Size.—As the pulmonary resonance is due to the air contained in the lung, diminution in the amount will be attended by a corresponding decrease of resonance. If the displacement of the air is complete, then percussion of that portion of the lung will be similar to that obtained over any solid, airless tissue, as the thigh or liver, and the sound will be designated as flat.

It is necessary that a clear distinction be made between dullness and flatness. As long as any resonance can be detected, the term dullness must be used with some qualifying word denoting its degree. Flatness means that the sound is absolutely devoid of any resonance.

It is rare for pulmonary consolidation to give a perfectly flat note, as there is usually enough air in the bronchi of the affected portion to give slight resonance.

Liquid effusions and solid growths in the pleural cavity, on the other hand, are frequently large enough to give a perfectly flat sound.

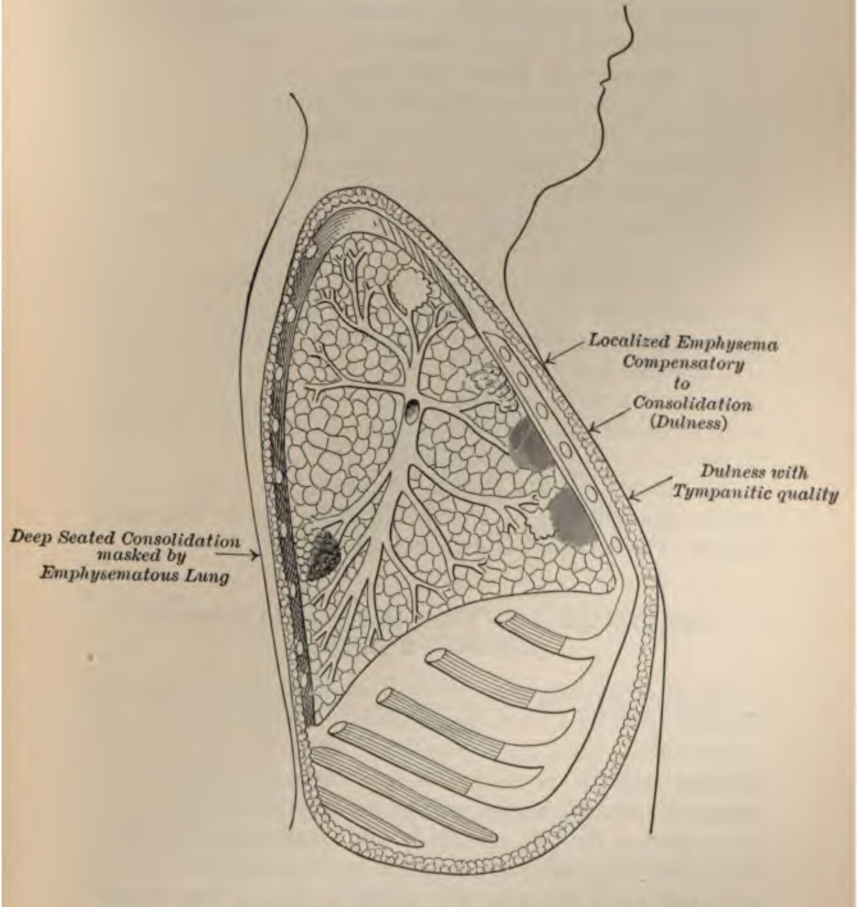
FIG. 19.



Changes in percussion note.

The percussion note obtained when the lung has been rendered more or less airless or solid depends upon the seat of the consolidation. If the consolidated area is close to the surface, immediately underneath the pleura, its influence upon the resonance would be marked, and

FIG. 20.



it can be best detected by light rather than by heavy percussion. If, on the other hand, it is situated deeper in the lung, and separated from the pleura and bony thorax by a zone of air-containing lung, then, in direct proportion to its depth, will it influence the resonance obtained, and more forcible percussion is needed. This can be best understood by referring to Figs. 19 and 20.

(5) **Influence of Heart and Liver.** — The resonance of the lung is impaired by the solid structures (heart and liver) that are covered to a greater or less extent by pulmonary tissue. The extent and degree of the dullness will depend upon the size and mobility of the lung on the one hand and that of the solid organs on the other.

The dullness that is detected over these organs, which gradually increases in degree until it merges into the perfectly flat sound of the organ itself, is known as relative cardiac and liver dullness. The influence of these organs upon the resonance and force of the blow necessary to elicit this relative dullness is indicated by Figs. 21 and 22.

The normal boundaries of these areas and the diagnostic significance of changes in extent are discussed under "Percussion," Parts III and IV.

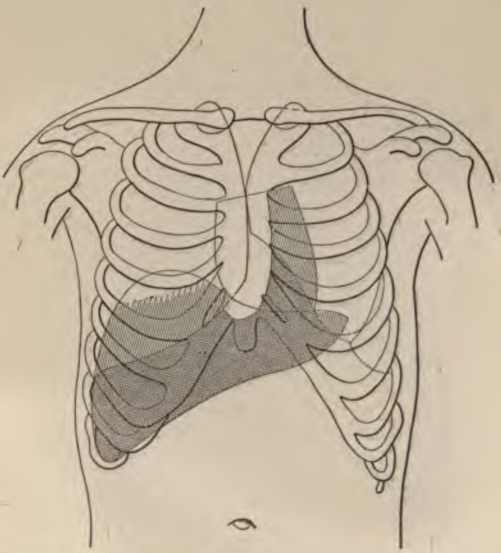
INDIVIDUAL AND REGIONAL VARIATIONS OF PERCUSSION SOUND.

From what has been said about influence of the different structures upon the percussion note, it is readily appreciated that no standard can be set. Each patient will have an individual variation within the range of normal, as the soft parts are thick or thin, the bony thorax flexible or rigid, the lungs large or small, with high or low tension, and, in addition, the sound will vary according to the portion of the thorax percussed.

As the value of percussion lies in the comparison of the note obtained over corresponding spots on the two sides, it is necessary to keep in mind the normal variations that occur over these spots.

The percussion note over the right lung, from the apex to the second interspace in the axillary line, is slightly higher in pitch, and duller than on the left side. This is due to the bronchi on the right side being relatively larger and nearer the surface. The change in percussion corresponds to the increase in vocal fremitus over the same area.

FIG. 21.



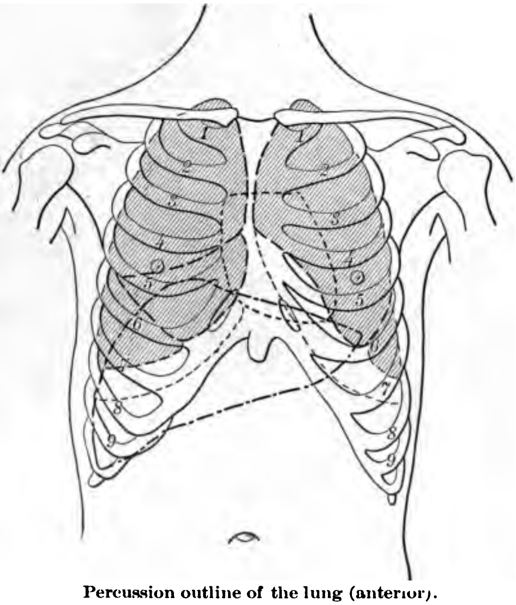
Absolute and relative heart and liver dullness, also regional variation in thorax.

The percussion note on the right side from the fourth interspace is modified by the relative liver dullness, while on the left side, beginning at the third interspace, close to the sternum, and corresponding to the site of the heart, the cardiac flatness and relative dullness is present. These variations are noted in Fig. 21.

PERCUSSION OUTLINES OF THE LUNG.

Percussion gives valuable information concerning the size and mobility of the lung, and no examination is complete unless the percussion outline of the borders of

FIG. 22.



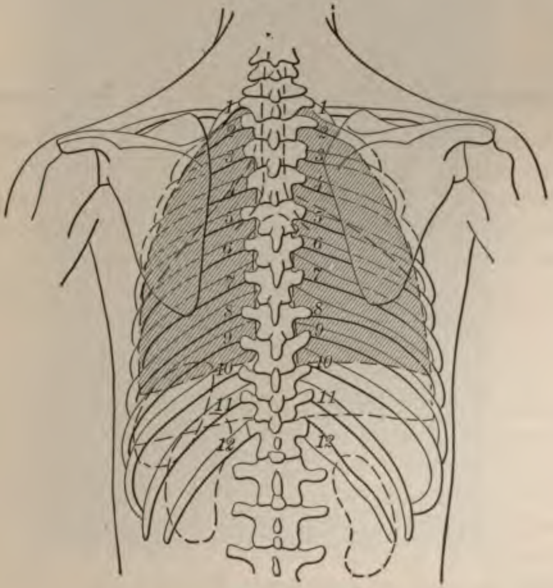
Percussion outline of the lung (anterior).

the lung during inspiration and expiration is determined.

The only boundaries or borders that can be definitely mapped out by percussion are a superior and inferior, and that portion of the anterior that is uncovered by the sternum and overlies the heart. At the end of quiet inspiration the superior border of the lung reaches two or three centimeters above the clavicle, occupying the supraclavicular space.

With full inspiration, the superior border is carried a finger to a finger and a half higher, and the space is filled out in all directions; while the percussion note is clearer and fuller from the increased tension of the pulmonary tissue and enlargement of the air spaces. The superior border does not rise as high when there is pleuritic thickening and adhesions. In emphysema the superior border is higher than normal with expiration, but there is not a corresponding increase in size during inspiration, and the note has a tympanitic quality, with but slight change at the end of full inspiration. The

FIG. 23.



Percussion outline of the lung (posterior).

inferior borders on the two sides are slightly different. On the right side, starting from the base of the xiphoid cartilage, it curves obliquely downward and outward across the sixth rib in the axillary line, the tenth rib in the scapular line, and meets the vertebral column at the junction of the eleventh rib.

On the left side, the inferior border can only be determined in the axillary line, as the presence of the stomach renders its detection in the mammary line difficult. (Figs. 22 and 23.)

AUSCULTATORY PERCUSSION (Stethoscopic Percussion)

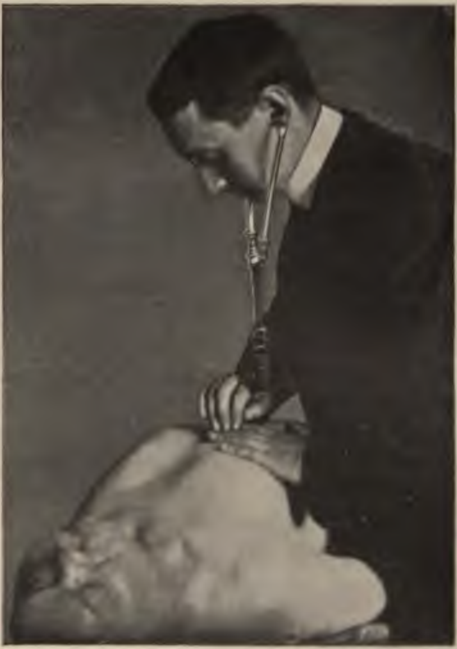
In this method the chest piece of the stethoscope is placed upon the chest over the organ that is under examination; mediate and immediate percussion is made from a distance toward the point where the stethoscope rests, and the variations in the sound are noted. When the percussion is made directly over the organ, a marked increase in the intensity of the vibrations is both heard and felt.

Within certain limitations, it is a valuable method of examination, allowing of a differentiation between conditions giving the same sound, as between the flatness of the heart and liver where the two organs are contiguous; between right-side pleurisy with effusion, pulmonary consolidation and liver flatness.

In order to be of use, the chest piece of the stethoscope should be small and rest wholly over the organ under examination. A special part to replace the ordinary chest piece should be used, so as to allow of its being placed between the ribs, thus avoiding the vibrations readily conveyed by the bony thorax. The percussion should be light, and, as the value is comparative, the points percussed should be equally distant from the tip of the stethoscope.

Another method which I have found very sensitive, and which avoids the vibrations of the bony thorax, is to listen to the percussion note with the chest piece of the stethoscope not touching the surface, but held just above and close to the point of percussion. In mapping out the relative dullness of the heart and liver, it is more definite than when chest piece rests on the bony thorax. (Fig. 24.)

FIG. 24.



Auscultatory percussion.

(A) Those which depend upon the variations in the normal sounds. These variations are limited to *(a)* quality or character, *(b)* intensity, *(c)* pitch, *(d)* duration, *(e)* rhythm, *(f)* the relative length of expiration and inspiration to each other.

(B) To the production of new or adventitious sounds or râles, which may be dry or moist, friction, etc. See page 112.

During inspiration enlargement of the thoracic cavity occurs in all directions, causing *(a)* movement of air in the respiratory tract; *(b)* change in the tension of the tissues forming the walls of the bronchi, bronchioles and alveoli.

In quiet breathing 30 cubic inches of tidal air are drawn into the respiratory tract during inspiration and forced out during expiration, and, as the estimated capacity of the larynx, trachea and bronchi is 10 cubic inches, and that of the alveoli after quiet expiration is 150 cubic inches, it is evident that the tidal air is carried even into the alveoli, and that there is aerial movement throughout the entire respiratory tract. Passing to and fro through the glottis, the tidal air is thrown into vibrations that are intense enough to be audible.

Laryngeal Breath Sounds.—The sound produced at the glottis is known as the laryngeal breath sound, and has the following characteristics: It is heard with both inspiration and expiration, but with a distinct break or pause between them, as the inspiratory sound is not audible during the latter part of the act.

The sounds produced during inspiration and expiration are nearly equal in length, that of expiration being slightly longer. The sound is harsh and blowing, the quality being tubular; the pitch is high, that of expiration being harsher and higher pitched than that of inspiration.

When the mouth is closed and the breathing is through the nose, the sound produced is more intense, harsher and higher pitched, as the vibrations made at the glottis are reinforced by those produced in the pharynx. The laryngeal sound may be imitated by placing the tongue in the position to pronounce "h" or "ch" and breathing deeply and regularly.

The intensity and character of the laryngeal sounds depend *(a)* upon the force and rapidity with which the tidal air rushes through the glottis; *(b)* the size of the glottis; *(c)* the position of the vocal cords; *(d)* the condition of the tissues of the larynx. Thus the sounds vary to a certain extent in each individual. As the vibrations made at the glottis are the basis of all sounds normally heard over the lung, the laryngeal breath sounds serve as a standard in each case by which an estimate of what should be the normal intensity of the respiratory sounds heard over the other portions of the chest.

The vibrations made at the glottis are conducted through the respiratory tract simultaneously by two channels: (1) Aerial, *i. e.,* vibrations of columns of air contained in the bronchi, bronchioles and alveoli; (2) the tissues of the walls of the bronchi, bronchioles and alveoli.

Aerial Conduction.—The laryngeal sound is conducted by the aerial vibrations through the trachea to its bifurcation into the primary bronchi almost unchanged, except for a slight toning down of the harshness. The laryngeal, or tracheal, sound represents the highest type of tubular or bronchial breathing, and is normally heard over the larynx and trachea; at times over the upper portion of the sternum in front and over the vertebral column as far as the fifth dorsal vertebra behind. It is heard over other regions of the chest only when pathological changes have occurred in the respiratory tract, that permit its conduction from its normal site to other areas.

At the bifurcation of the trachea, the column of moving and vibrating air is divided into two uneven parts, the larger passing to the right bronchus, the smaller to the left. With this division certain changes occur, which influence the aerial vibrations that have been carried down from above.

(1) The united capacity of the two bronchi being greater than that of the trachea, the force of the current of tidal air is less.

(2) Reflection, or reverberation of the laryngeal vibration, which causes confusion of the sound.

(3) The impinging of the column of air on the angle of bifurcation adds new vibrations and a new quality of sound to that received from above.

Tissue Conduction. — The vibrations transmitted through the walls of the trachea and bronchi convey the laryngeal sound unchanged in quality, and but slightly weakened.

The effect of these two series of vibrations is to so modify the sound in the primary bronchi that it can be distinguished as a type, *i. e.,* bronchial breathing.

Bronchial breathing differs from the laryngeal sound as follows: The tubular quality is slightly diminished, and is less sharp, the change being most marked on inspiration. The pitch is not as high, and there is a corresponding loss in intensity and harshness. Inspiration and expiration are nearly equal in duration, expiration alone being slightly shortened, and the pause, while present, is not so marked.

As the tidal air descends along the bronchial tract, at each division of the column similar changes occur, as at the bifurcation of the trachea.

THE MODIFICATIONS OF THE AERIAL VIBRATIONS that occur in the different divisions of the bronchi are as follows: (1) The increased capacity of the branching tubes *(a)* reduces the force of the current in the tidal air and *(b)* causes diffusion of the vibration. These two factors lead to a loss of intensity in the sound produced.

(2) The refraction or reverberation occurring in the

bronchi causes the tubular laryngeal sound to become confused in character, the harsher tones being especially modified, while the more musical elements persist. This makes the pitch of the sound lower than that which is heard above the large bronchi and larynx.

Obliteration or change in the harsher qualities has been variously explained, but the mere fact of their loudness or harshness causes their reverberation to occur more easily, and therefore the primary vibrations are interfered with by the secondary vibrations of reverberation.

(3) At each bifurcation of the bronchi the moving column of tidal air has sufficient force to set up new vibrations, which, being added to those received from above, modify the quality of the sounds; and although the sound remains tubular, it has a softer blowing character.

THE MODIFICATIONS OF VIBRATIONS THROUGH TISSUE CONDUCTION.—The walls of the large and medium-sized and cartilaginous bronchi are good conductors of vibration. The change that occurs as the vibrations are carried through the tissues of the different branches of the bronchial tree is chiefly loss of intensity, but the bronchial character persists. On account of the rigidity of the bronchi, tissue vibrations are influenced but slightly by the aerial vibrations within them.

As a result of these two classes of vibrations, aerial and tissue, the sounds heard over all portions of the bronchial tract will be more or less characteristic. The pause between the inspiratory and expiratory sounds in the larynx is well marked. This pause becomes less and less marked the farther down the respiratory tract the examination is made. This is due to the more continuous movement of air during inspiration and expiration in the lower divisions of the bronchi. Above there is merely the tidal air going in and out; below there is the more continuous movement of not only the tidal air, but also the columns of residual air. During the latter portion of inspiration the air passes through the glottis without setting up audible vibrations, while, on the other hand, on account of the position of the vocal cords during expiration, audible vibrations are produced throughout the entire act. Lower down in the bronchi the added vibrations cause a continuous sound, increasing in intensity, to be made throughout inspiration. While, on the other hand, in expiration the current becomes progressively feebler.

In the larynx and large bronchi the tubular quality of the sound is equal during inspiration and expiration. Over each successive lower division of the bronchi the inspiratory sounds lose their pure bronchial character, while the expiratory sound retains it to a greater degree. This difference in the amount of tubular element is due to the fact that in inspiration new vibrations are added at each division of the bronchi, which modify the tubular quality. During expiration, on the other hand, no new sounds are added.

If it were possible to apply the ear or the stethoscope to the different subdivisions of the bronchial tree, each would give its own peculiar type, having a characteristic quality, pitch, intensity and duration. (Fig. 25.)

Certain pathological conditions allow these varying degrees of bronchial breathing to be heard at the surface, and according to the character of the sound heard the nature and extent of the anatomical changes in the lung are determined.

Changes in the Bronchioles and Alveoli.—From what has already been said of conduction of the glottic vibrations by the air contained in the trachea and bronchi, and by the walls of the tubes, and the modifications that occur in the sound in different portions of the bronchial tract, it is easy to understand that still greater modifications will take place in the bronchioles and alveoli. As long as the divisions of the bronchial tubes contain cartilage and muscular tissue, the vibrations transmitted through the homogeneous tissue preserve in a large measure the tubular quality of the laryngeal sound, and are not easily modified by the aerial vibrations.

"Below the diameter of 1-24 inch (1 mm.) the bronchial tubes have neither cartilages, mucous glands nor any continuous muscular coat; circular and longitudinal elastic fibres replace the muscular coat." (Powell.)

This change in the structure of the bronchi has a marked influence on the conduction of the laryngeal sounds onward towards the surface, which has been so marked a feature of the vibrations transmitted by the tissues. As the structure of the bronchioles and alveoli becomes less and less homogeneous with that of the larger bronchi, the vibrations received from above are not conveyed so easily. The loss of the tissue vibration causes a dampening of the tubular or bronchial quality of the sound. As the structures of the bronchial tubes and alveoli becomes more membranous and tense, they are influenced to a greater degree by the aerial vibrations.

The rapid breaking up of the smaller bronchi into bronchioles causes a still more marked change in the aerial vibrations received from above. They are diffused, reflected again and again; the sound is confused; the harsh tubular and blowing character is lost; the softer, more continuous and rustling (musical) quality alone remaining.

There has been much discussion whether the movement of the tidal air in the bronchioles and its passage into the alveoli is sufficient to produce audible vibrations, and whether the sound heard at the surface of the lung during inspiration is due entirely to the vibrations made at the glottis, changed by transmission through the lung, or is in part composed of vibrations made at the junction of the bronchioles with their infundibuli. Some claim that the sound heard at the surface during inspiration is made in the alveoli.

After quiet expiration, the capacity of the alveoli is estimated at 150 cubic inches; the amount of tidal air in quiet breathing is 30 cubic inches, which in forced inspiration and expiration may reach 200 cubic inches in man and 150 cubic inches in the female. There is no question that it is possible for the tidal air to pass from the narrow bronchioles into their relatively wide infundibuli, and to produce vibrations which, if not audible in themselves, would intensify in some respects and modify in others the vibrations, already present, which had their origin at the glottis.

The aerial vibrations, and especially those in the alveoli, play an important part in the production of the inspiratory sound heard at the surface. They explain why the vesicular element is present to a greater or less extent as long as tidal air reaches the alveoli, even though changes in the stroma of the lung may allow bronchial sound to be heard through tissue conduction, and why inspiration does not have as tubular a quality as expiration. In inspiration through the enlargement of the thoracic cavity the tissues of the alveoli and bronchioles are rendered more tense, have greater power of conduction, and are more easily thrown into vibrations, so that there is a corresponding increase in the intensity of the sound up to the end of the act.

Normal Respiratory Murmur.

Over the surface of the lung the breath sounds (the respiratory murmur) have the following characteristics or elements during inspiration:

The *quality* is variously described as breezy, rustling, sighing, slightly shuffling or vesicular, corresponding to the sound produced by regular and natural breathing when the lips are placed in a position to pronounce "F" or "V." The *pitch* is low; the *intensity* increases to the end, and is heard throughout the act *(duration)*. The *rhythm* is regular. The sounds of inspiration and expiration occur at regular intervals.

All these elements of the respiratory murmur will vary normally within wide limits in different individuals, but will always have a definite relation to the intensity of the sounds heard over the larynx and trachea, as has already been noted.

The expiratory portion of the respiratory murmur immediately follows that of inspiration, and differs in the following respects: The quality is harder, the breezy vesicular element being replaced by a slight blowing sound; the pitch is higher;[1] the intensity is less, being loudest at the beginning and rapidly fading out, so that its duration is only one-half to one-third that of inspiration.

It is necessary to explain why these differences occur in expiration in order to understand the occurrence of pathological changes.

It has already been noted that inspiration is a muscular act which continues until the thorax is fully expanded. The laryngeal sound is made at the glottis, and is conducted downwards by, (1) the current of tidal air which is towards the surface of the chest, and (2) by the tissues in the bronchi, bronchioles and alveoli, the tension of which increases to the end of the act. In addition to the vibrations made at the glottis, other vibrations are added at different parts of the tract, and especially at the opening of the bronchioles into their infundibuli. All of these factors cause the inspiratory part of the murmur to have its peculiar quality and pitch; to increase in intensity to the end, and to be heard throughout the act.

Expiration, on the other hand, is normally a passive act, which depends upon the elasticity of the thorax and the pulmonary tissue and the contraction of the muscle fibres present in the bronchi. This force is greatest at the beginning. The sound in the larynx is made at the level of the vocal cords, and the direction of the tidal air is upwards and away from the surface of the chest. The direction of the expiratory current of air interferes with aerial conduction of the sound downwards. As the tidal air is forced out of the alveoli into the tubes, no new vibrations are made in the bronchioles or tubes.

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[1] Many authors state that the pitch of expiration is lower than that of inspiration.

The sound that is heard at the surface in expiration is chiefly that conveyed by the tissues of the respiratory tract, so that the quality lacks the vesicular element and is somewhat blowing and hard, and its pitch is higher than inspiration sound. As the tidal air is forced out of the lung the tension becomes less, losing the power to conduct, so that the intensity is most marked at the beginning, rapidly diminishing; and the sound is heard only during a short part of the time, or may be entirely wanting, especially where the expiratory laryngeal sound is naturally very soft or the expiratory force weak.

The fact that during expiration the vibrations are chiefly conducted by the tissues explains the tendency of the blowing or bronchial element to be so much more pronounced during this portion of the murmur.

REGIONAL VARIATIONS OF THE BREATH SOUNDS.—The breath sounds vary in intensity, duration, quality, pitch and in the relative lengths of the sounds heard during inspiration and expiration over different portions of the lung.

It is necessary to have a thorough knowledge of the normal variations in order to recognize the alterations that occur in morbid conditions, because a type of breathing that is normal for one region would be pathological if heard in another.

It has already been shown that over the larynx, trachea and primary bronchi the tubular sound is heard unmodified, and constitutes the laryngeal, tracheal and bronchial types of breathing.

Over the interscapular space, opposite the spine of the scapula, the sound is neither pure bronchial nor entirely vesicular in quality and pitch, but is of an intermediate character. The tubular sound is modified, so that inspiration has a soft, blowing, but somewhat bronchial, character, and the pitch is not as high as over the trachea. The expiratory sound is separated from the inspiratory by a shorter pause, and corresponds to the inspiration sound in quality and pitch, but its duration is not as long as over the trachea. The tubular element is more marked on right side than the left, on account of the relation of the trachea and bronchi to spine and ribs. (Plate II.)

In front of the chest on the right side, at the junction of the second rib with its costal cartilage, the respiratory murmur differs from that heard on the left side in the corresponding region. While vesicular in character, there is a slight blowing element added, and its intensity and pitch are slightly raised. Over the right apex of the lung the same difference is present in a more limited degree, due to the origin of the bronchus supplying the upper lobe of the right lung.

The reason for the normal regional differences of vesicular murmur is the relative nearness of the large bronchi to the chest wall, so that, although these are covered by lung tissue, the elements of the bronchial sound are present in the respiratory murmur in a greater degree than elesewhere.

Types of Respiratory Sound.—Various classifications have been made of the different types of breath sounds heard in health and in disease.

Some authors use as a basis of their nomenclature the diseases of which the sounds are diagnostic; others employ terms descriptive of the pathological changes. The one that is most commonly used, and also the most satisfactory, is that which considers the elements of the sounds and groups the types according to the changes that occur in intensity, rhythm, duration, quality, pitch and the relative length of inspiratory to the expiratory sound.

CHANGES IN INTENSITY. *(1) Increased, Exaggerated, Compensatory, Vicarious, Puerile Breathing.*—The chief change from the normal is in the intensity of the breath sounds. Inspiration has the vesicular element accentuated. Expiration is slightly longer than normal, and of a soft, blowing character. It occurs whenever the breathing is very deep and active, as in forced voluntary breathing; also in dyspnœa and over portions of the lung, which by increased activity are compensating for imperfect action in another part.

Puerile breathing, which is the normal breathing of children under twelve years of age, differs from exaggerated breathing, in that both inspiration and expiration are abnormally loud and sharp, and expiration is nearly as intense and long as inspiration. It is somewhat blowing in character, and may have a slight bronchial quality.

(2) Diminished, Feeble, Senile, Emphysematous Breathing.—The sounds are faint, and heard only for a short time, and expiration may be inaudible. The diminished intensity may be due to shallow breathing, imperfect muscular action caused by weakness, pain in the muscles, nerves or pleura; rigidity of the bony thorax; thickness of the soft parts of the thorax and thickenings of, or exudations and effusions into, the pleural sac; interference with the movement of air through the bronchi; weak glottic sound, dependent upon normal or pathological conditions of the larynx.

The breath sounds that occur when the elasticity of the lung is impaired, as in emphysema and old age, differ from simple diminished breath sounds, in that the inspiratory is short and weak, and the expiratory sound is relatively stronger and longer.

(3) Absent or Suppressed Breathing.—The breath sounds are not heard. This is due to changes that render conduction to the surface impossible. It is most marked in effusions of serum, pus or blood into the pleural sacs; in closed pneumo-thorax with pulmonary

collapse; over cavities filled with fluid; with occluded bronchus, and occasionally when there is great thickness of the chest wall associated with feeble breathing.

ALTERATIONS IN RHYTHM.—*Interrupted, jerky, wavy, cog-wheel breathing.* Instead of the inspiratory breath sound being even and continuous, with a gradual increase in intensity, it is broken and puffy. The expiratory sound may also be uneven.

This may be due to irregular muscular action, as in nervous patients and in painful conditions of the thorax and pleura, or to interference with the passage of air through the bronchi, as occurs in early tubercular infiltration of the apices, "when the air enters different lobules at different times."

Over the heart and large blood-vessels a peculiar, jerky, puffing sound may be heard, due to the cardiac systole forcing from the alveoli.

ALTERATIONS IN DURATION.—The significance of change in duration of breath sounds depends upon *(a)* whether the normal relation is preserved between the sounds of inspiration and expiration; *(b)* whether it is associated with adventitious sounds, and *(c)* whether there is any alteration in pitch and quality.

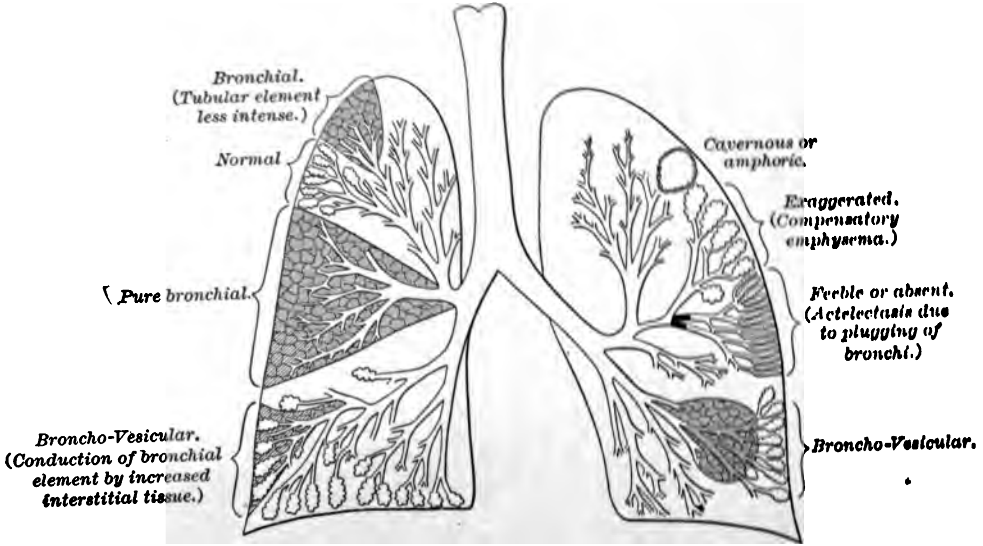
The inspiratory and expiratory sounds, while preserving their normal relations, may be lengthened or shortened by all conditions which have been mentioned as producing increased or diminished breath sounds.

Prolonged Expiratory Breath Sound.—This occurs under various conditions.

(1) When the expiratory sound is prolonged, but is low pitched and faintly blowing in quality, it depends upon diminished elasticity of the lungs, and is associated with decrease in the length of inspiratory sound, with a slight pause between inspiration and expiration. Loss of elasticity occurs in old age and in emphysema. This type of breathing has therefore been designated as "emphysematous."

(2) The expiratory sound may be prolonged, and

FIG. 25.


Bronchial. (Tubular element less intense.)
Normal
Pure bronchial.
Broncho-Vesicular. (Conduction of bronchial element by increased interstitial tissue.)
Cavernous or amphoric.
Exaggerated. (Compensatory emphysema.)
Feeble or absent. (Actelectasis due to plugging of bronchi.)
Broncho-Vesicular.

Diagramatic illustration of modification of breath sounds.

accompanied by moist or dry râles, due to narrowing of the tubes by secretion or by spasm of the muscular tissue, as occurs in asthma, bronchitis and the early stage of pulmonary tuberculosis.

(3) A prolonged expiratory sound may be associated with a higher pitch and a blowing quality, due to changes in the lung which increase its power of conducting to the surface the sound which is normally present in the bronchi throughout the entire expiratory act. This may be due to increase in or induration of the normal structure of the lung, as in the early stages of tubercular infiltration, interstitial pneumonia or fibroid thickening.

This type of breathing is normally present over the apex of the right lung.

A prolonged expiration sound, associated with slight increase of the tubular quality and heightening of the pitch, marks the faintest alteration in the elements of the respiratory sounds in the transition from the normal vesicular murmur to the different degrees of bronchial breathing.

ALTERATIONS IN QUALITY AND PITCH.—The changes that occur in the quality or character of the breath sounds depend upon the addition of the bronchial and tubular and decrease of the vesicular elements. The extent to which this may occur varies. Based on the proportion of the vesicular and bronchial quality present, various subdivisions of bronchial breathing have been made:

(1) Broncho-vesicular, indeterminate, subtubular, hinted or indistinct bronchial breathing. Sharp breathing with bronchial quality in expiration; transition breathing; rude respiration; harsh breathing.

As the names indicate, the distinguishing feature of this type is the presence of both vesicular and bronchial quality. This depends upon the changes in the lung, which allow the bronchial sound to be heard at the surface. These changes may be due to tubercular infiltration, croupous pneumonia, or fibroid induration from any cause. It is normally present in the interscapular region.

The term "broncho-vesicular" is by some extended to all cases of bronchial breathing which do not have the well-marked tracheal quality, while others limit it to that type where a faint bronchial quality is added to the vesicular breathing.

The terms "rude" and "harsh" have been used by some authors to describe certain grades of broncho-vesicular breathing, but should be discarded, as they are misleading, having been employed by others as synonyms for exaggerated breathing.

(2) Pure bronchial breathing is characterized by absence of the vesicular quality. Both the inspiratory and expiratory sound are high pitched and tubular; that of expiration the more markedly so.

(3) High-pitched, blowing, tubular and tracheal breath sounds merely have reference to the intensity and extent of the bronchial elements.

(4) Cavernous and amphoric breathing are modifications of the bronchial type. The former is low pitched and hollow; the latter is high pitched and metallic.

(B) ADVENTITIOUS SOUNDS.

In addition to the breath sounds present in the chest, there may be heard certain abnormal or adventitious sounds, which depend entirely upon pathological changes in the bronchi, alveoli and pleura.

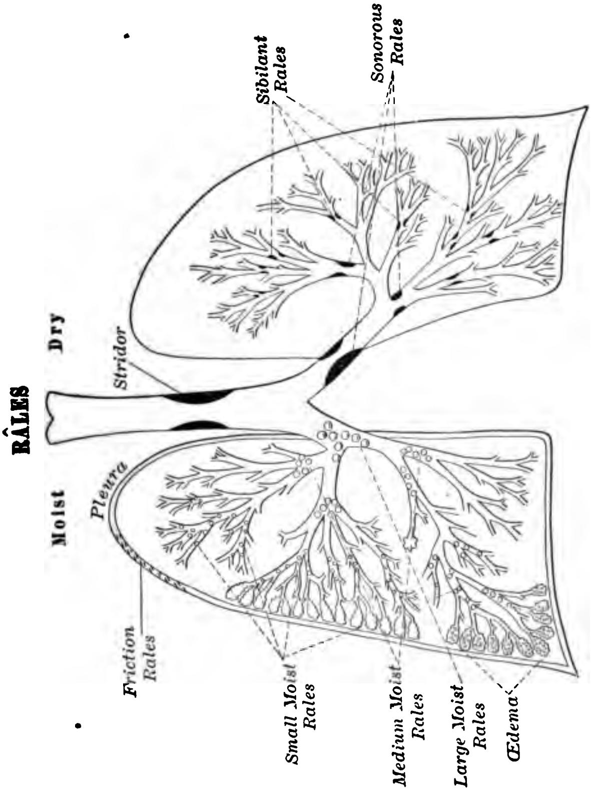
Adventitious sounds are divided into three classes:

Dry Râles, Moist Râles and Friction Sounds.

(1) **Dry Râles,** or rhonchi, are musical sounds, produced by vibrations set up in those bronchial tubes whose lumen has been markedly narrowed by tenacious

exudates, contraction of the muscular coats or pressure of tumors. These changes are of such a nature as to practically convert the bronchial tubes into wind instruments.

FIG. 26.



Diagramatic illustration of dry and moist râles.

These sounds or râles may vary in quality, pitch, intensity and duration, according to the size of the tubes involved.

They are usually divided into two classes, sonorous and sibilant.

Sonorous râles are snoring, low-pitched, loud sounds, which may be heard during both inspiration and expiration, or may be limited to one part only of the respiratory act, according to the nature of the cause. They may be heard in connection with the vesicular murmur, or may entirely obliterate it.

Stridor is a type of sonorous râle in which the vibrations are very coarse, and the sound produced hoarse and low pitched. It occurs in diseases of the larynx, trachea and main bronchi. The vibrations producing stridor and sonorous râles may be strong enough to produce tactile fremitus, as described in palpation.

When the narrowing in the trachea and large bronchi is not sufficient to produce a musical sound, but is still sufficient to increase the tubular quality normally present, it will influence the respiratory murmur by adding a bronchial element, as will be described later.

Sibilant râles are whistling, piping, squeaking, humming, hissing sounds, which are markedly musical, shrill and high pitched. The quality and the pitch will depend upon the size of the tube, the degree of contraction, the force of the current of air, the conditions of the pulmonary tissue and the conduction of the sound to the surface.

These râles are permanent or transient, according to the nature of the cause. When the narrowing of the tube is due to pressure of a tumor or to a growth within the bronchus, the râles will be permanent. When, on the other hand, the sound is caused by secretions in the tubes or spasm of the muscular fibers, they are variable and inconstant, disappearing from one spot and appearing in another.

They also change in character. At one time they may be hissing, shrill or whistling, and again sonorous, as tubes of a different size are involved. Coughing, deep inspiration or the inhalation of chloroform or ether will modify those of a transient nature, according to the cause.

While usually there is little difficulty in determining what râles belong to the dry type, in the finer, less musical sibilant ones it is often a matter of opinion how they are to be classified.

(2) **Small, Medium and Large Moist Râles.** — MOIST RÂLES, as the name indicates, are due to the vibrations produced by the movement of air through fluid (mucus, serum, blood or pus) in the air-containing spaces of the lung.

Certain characteristics of the râles depend upon the nature of the fluid. When it is thin and watery, the râles will have a moist, bubbling sound; when it is thick, in addition to the moist sound, there will be an element of sharpness or stickiness, as the bubbles burst with an explosive noise.

The quality and pitch of moist râles are modified by the condition of the surrounding lung. When the lung is normal, the sound of the râle loses its sharpness in transmission, the moist character only remaining. But in consolidated areas the râle has a certain ringing character, due to the supposed resonating effect of the consolidated structures and their increased conducting power.

The variations that occur in the characters of moist râles have led writers and teachers to adopt various classifications and to designate their subdivisions by names which they considered more or less descriptive of the sound, or of the pathological condition that rendered their occurrence possible.

Unfortunately, different authors do not use the same descriptive terms in the same sense, so the "crepitant" râle includes not only the finest crackling sound, but also those of larger size, which are by others classified as "subcrepitant," or "small mucus."

This want of agreement in classification and nomenclature has led to great confusion, and it is particularly perplexing to the student, as it conveys the impression that there is a diversity of opinion as to the diagnostic significance of these adventitious sounds.

It is almost hopeless to arrange a classification that will include all the terms used.

Fortunately, the tendency is more and more to avoid a specific descriptive term, and to describe the sound in terms of quality, pitch, duration and intensity.

CREPITATIONS, OR "CREPITANT RÂLES."—These are the minutest of the moist râles, and have been by some writers described as belonging to the dry type. They have a fine, somewhat dry, crackling sound, similar to that produced by slowly and firmly rubbing a lock of hair between the finger and thumb close to the ear. At times they have a sharpness that resembles the sounds produced by the crackling of salt thrown upon the fire, or the sound gives an impression of stickiness, as when the moistened thumb and finger, having been pressed together, are separated close to the ear.

Their pitch is high, and they convey the impression of being produced near the surface. They occur in showers or explosions, and are of uniform size.

They are persistent at the spot first heard, not being removed by coughing; and are usually restricted to inspiration, although they may at times be heard momentarily just at the beginning of expiration. Some authors state that they may be heard only during expiration. Formerly it was claimed that one of the distinguishing features of the crepitant râle was that it occurred only during inspiration, and that any râle having the same general characteristics, but occurring during expiration, could not be a crepitant râle.

There are differences of opinion as to the place and mode of production and diagnostic significance of the crepitant râle. Some claim that they are intrapulmonary, being produced in the ultimate bronchioles and alveoli, and due to the separation of the walls of the alveoli, which were more or less firmly agglutinated by tenacious secretions. Others claim they are caused by the bursting of fine bubbles, formed by the forcing out, during inspiration, of the secretions from the bronchioles into their infundibuli. Others believe that they are extrapulmonary, and are made in the pleura, being, in fact, the finest of friction sounds.

The facts that seem to uphold the latter theory are their remaining at the spot where first heard, not being influenced by coughing; that they are usually heard only with inspiration, but may occur with expiration; and that a forcible pressure by the ear or stethoscope upon the chest wall during expiration will often cause them to be heard with expiration, when before they were only detected with inspiration. While production of the crepitant râles in the bronchioles and alveoli is possible, their pleural origin is more probable.

These râles have a special importance, as they have been considered diagnostic of the first stage of croupous pneumonia, occurring even before changes in the respiratory murmur. In this disease they have also been called the "râle indux," and confusion has occurred by considering them also the "râle redux."

They occur also over the seat of catarrhal pneumonia, caseous tubercular pneumonia, infarctions, œdema of the lung and atelectasis.

Dry crepitations are frequently heard at the base of the lung in persons who have been long confined to the recumbent posture, and in those in whom there has been superficial breathing. They are most apt to be heard after the fortieth year, and disappear after two or three full breaths have been taken. These râles are caused by the action of lung that has been for some time in the quiescent state.

Classification of Moist Râles.—The most rational division of the moist râles is, according to their size, into small, medium and large râles.

Appreciation of the size of a râle is important, as it indicates *(a)* the size of the bronchus in which the sound is made; *(b)* it is diagnostic of the extent of the disease, and whether or not further pathological changes have occurred beyond the mere presence of more or less fluid secretion in the tubes.

The size of the râle should always be considered in conjunction with its intensity and nearness to the ear. The larger the râle, the larger should be the space in which it is made, and consequently it should give the impression of being more or less distant from the surface.

If, on the other hand, there is heard over a region where the bronchi are normally small, as at the apex or base, fairly large-sized râles, but at the same time appearing to be close to the surface, it indicates that there has occurred dilatation of the bronchi (bronchiectasis), or destruction of pulmonary tissue, and the formation of cavities of greater or less extent.

The small moist râle, in addition to its size, differs from crepitations or the "crepitant râle" in the following: *(a)* The liquid character is more marked. *(b)* While heard chiefly with inspiration, it is also present during the first part of expiration, and may persist throughout. *(c)* It is influenced by coughing, at times disappearing when the bronchi are clear of their fluid contents; at other times appearing when the secretion is forced from the alveoli into the bronchi.

The designation by some authors of these relatively large moist râles as "crepitant" has led to much confusion.

The small moist râles are divided into two groups, according to the quality of their sound.

While both groups of the small moist râles are due to the presence of fluid secretion in the air-containing spaces, the difference in the quality of sound is dependent upon the condition of the pulmonary tissue, its influence on the conduction of the râle sound to the surface, and on its power to modify.

(1) When the pulmonary tissue is normal, the moist

sound made in the bronchi is modified, as is the bronchial sound that is normally present at the site where the râles are made, and they are heard at the surface as soft liquid or bubbling sounds.

Authors have used the terms "bubbling," "liquid," "mucous," "submucous," "non-ringing," "non-consonating" ("klanglos") to describe the sound.

(2) When there are present changes which increase the power of conduction by the tissues, or produce a resonating effect, as occurs in increased tension (children), or in pulmonary consolidation due to increase of tissue, or the filling of the air spaces by solid exudate (pneumonia, tuberculosis, etc.), the râles will be heard at the surface with increased sharpness. They will have a higher pitch and a crackling, explosive or ringing quality.

Hence the names "dry," "crackling," "crepitating moist râle," "humid crackling," "explosive moist râle," "subcrepitant," "ringing,""consonating" ("klingend"), given to describe this character of sound.

Râles in group 2 are usually associated with percussion, palpation and auscultatory signs that indicate the pathological conditions.

When this type of râle is not attended by these confirmatory signs, the quality is due to the tenacious character of the secretions.

The medium and large-sized râles are also divided into two groups, and present the same general characters as the small, but are larger-sized and are heard during expiration to the same extent as during inspiration.

The largest of the moist râles have received several names to denote their association with certain pathological conditions.

GURGLES are produced in cavities partially filled with fluid, where the bronchus enters below the level of the fluid. They may be present during inspiration and expiration, or may be induced only by coughing. They are large, liquid, bubbling sounds, with a peculiar hollow ringing quality, which distinguishes them from other moist râles of the same size.

MUCOUS CLICKS are sounds which may be imitated by whispering the word "click." They generally occur singly, and are short, snappy, sticky sounds, heard only during inspiration. Their mode of production is still in doubt, but they are generally associated with softening of tubercular deposits. This term is gradually falling into disuse, as these râles are not considered to have the diagnostic significance which was formerly given them.

METALLIC TINKLE is a single loud, high-pitched sound, with a marked echo quality, and is produced in large cavities with thin, tense walls. The peculiar echo quality of the sound is due to reflection or reverberation in the cavity. While heard with greatest distinctness in pyopneumothorax, it may occur over a large pulmonary cavity, when it is called "amphoric echo."

SUCCUSSION sound is a splashing sound that occurs when the pleural cavity contains air and fluid.

This sign may be induced by giving the patient a quick, short shake, or by coughing. While the sign is present in pneumo-hydro- and pneumo-pyothorax, it may occur in cavities of the lung when of large size and filled with fluid.

(3) **Friction Sounds.**—In normal conditions the movement of the two surfaces of the pleura on each other is unattended with any sound, but in diseased conditions râles of various kinds may be produced.

(a) DRY FRICTIONS OR CREPITATIONS.—When the surface becomes abnormally dry, the to and fro movements are attended with a dry rubbing sound, similar to that produced when the dry surfaces of the hands are passed lightly over each other, or it may be like the crepitations described above under moist râles.

Dryness of the pleura occurs during the first stage of its acute inflammation and when a large amount of fluid has been lost from the body, as in excessive diarrhœa of the choleraic type.

(b) Moist Crepitations.—When the pleuræ are covered with a tenacious secretion which glues the two surfaces together, their separation is accompanied by sticky, moist, crackling, crepitating râles. This type frequently replaces the dry râles, as the plastic exudation is poured out after the first stage of inflammation.

(c) Rubbing, Rasping, Grating, Grazing, Creaking, Leathery Sounds.—These occur when the surfaces of the pleura are roughened by exudation, by inflammatory changes or joined together by bands of adhesions. These râles may be felt by the patient, or detected by the hand or ear as fremitus.

As the pleural râles depend upon the movement of the thorax, they will be heard with the greatest intensity over the lower segment of the thorax at the end of inspiration and at the beginning of expiration, as the play of the pleura is greatest during these portions of the respiratory act.

When due to a sticky exudation, deep breathing frequently causes the râles to disappear. They can be heard again after quiet breathing and when pressure is made on the chest wall so as to increase the friction.

Differentiation of Pleural from Bronchial Râles.

In certain cases it is impossible, by the quality or size of the râle, to separate the pleuritic from the bronchial râles.

Pleural.	*Bronchial.*
Localized.	Not generally localized, but if so, they are associated with signs of bronchitis, or consolidation.
Generally unilateral.	Very apt to be bilateral.
Frequently accompanied by pain.	Generally no pain.
They give the impression of being produced close to the ear.	They seem more or less distant from the surface.

Usually increased by coughing or deep breathing.	Frequently disappear after coughing or deep breathing.
They are localized at the point where first heard.	The location changes frequently as the result of coughing.
Their intensity may be increased by pressure upon the surface.	Unmodified by such pressure.
Occur in showers or bunches, and all of uniform size.	Various sizes may be heard at the same time.

It must be borne in mind that pleuritic and bronchial râles may be present at the same time.

(4) **Indeterminate, or Indefinite Râles.**—In addition to the dry, moist and friction râles, another class has been recognized under the title of "indeterminate" or "indefinite" râles. They are crepitating, crumpling, crackling sounds, moist or dry, heard over various portions of the thorax during inspiration, expiration, or both. The origin and mode of production of their sound are doubtful.

They include the muscle sounds and the so-called emphysematous crackle. Some authors include in this class all râles that cannot be definitely determined as bronchial or pleuritic in origin.

CHAPTER VI.

AUSCULTATION (CONTINUED)

(A) CONDITIONS MODIFYING AERIAL VIBRATIONS AND CONDUCTION OF SOUND BY THE COLUMN OF AIR.

Movement of Tidal Air.

As the respiratory sounds depend primarily upon the movement of air through the glottis and bronchial tract, they will be influenced by the amount of tidal air, the force of the current, and the rapidity of its movement. These factors will influence chiefly the vesicular quality of inspiratory sound and the intensity and duration of both respiratory sounds.

When the breathing is deep, and at the same time more rapid than normal, the inspiratory sound is sharper in quality and more intense, while the expiratory has a more blowing quality, and its intensity is accentuated. When the muscles of expiration aid the normal elasticity of the lungs, the duration of the expiratory sound may nearly equal that of the inspiratory. When this type of breathing is general, it is called exaggerated breathing.

When the respiratory function of one lung is interfered with from any cause, as pneumonia, pleurisy with effusion, etc., and the unaffected lung is doing more work in consequence, the breathing over the unaffected side is called compensatory breathing.

Exaggerated or *compensatory* breath sounds may be limited to one lobe, as when pneumonia affects the other. They may be limited to a portion of a lobe. This occurs *(a)* when a bronchus is plugged, or *(b)* when some of the lobules are filled with inflammatory exudate. In such cases the unaffected lobules on this division of the bronchial tree accommodate not only their own normal quantity of tidal air, but also that which should go to the affected part; and not only is the current stronger, but there is overdistension of the alveoli, increased tension and resultant change in the vesicular murmur.

Localized exaggerated breathing in any portion of the lung always indicates compensatory action, and is therefore an important diagnostic sign.

Conditions which diminish the amount of tidal air, weaken its force, or slow the current, will cause a diminution of the breath sounds heard at the surface.

These conditions may be due to changes *(a)* in the bronchial tract, as in certain stages of bronchitis; *(b)* in the parenchyma of the lung, as in emphysema; *(c)* in the pleura, as adhesions or fluid; *(d)* in the bony thorax, as increased rigidity; *(e)* in the soft parts, as feeble muscular action.

In some individuals the normal vesicular murmur is so faint during quiet breathing that it can scarcely be appreciated. This may be due to the manner of breathing or to anatomical conditions of the larynx. In such cases it is necessary for the patient to take deep breaths in order to obtain the breath sounds.

Influence of the Larynx.

As has already been noted (page 99), the normal vesicular murmur will vary within wide limits, but will always have a definite relation to the character and intensity of the sound heard over the glottis, which forms the basis of all breath sounds present in the lung; so that as each voice has its own individual quality by which it is recognized, so also will the character of the respiratory sounds differ. Therefore, when the question arises whether or not the breath sounds are within the range of normal, the laryngeal sound should always be taken as a guide.

Morbid conditions of the larynx will modify the breath sounds in either inspiration or expiration, or in both.

As has been shown, the *inspiratory* portion of the vesicular murmur is composed not only of sounds made in the larynx, but also of those made in other portions of the bronchial tract and at the alveoli, while the expiratory portion depends entirely on the conduction to the surface of the sound made at the larynx. Therefore, any changes made in the character or intensity of the glottic sound will be most marked in the *expiratory* murmur.

The laryngeal sounds may be increased by anything which narrows the opening or changes the relation of the parts. The change in the breath sounds produced may be limited to one or both portions of the respiratory act. Narrowing of the glottis, depending upon malignant or non-malignant growth, tuberculosis and syphilis, may cause greater or less change in the quality of the sound, so that the respiratory murmur heard at the surface of the chest will have a peculiar harsh, blowing quality. Paralysis of the vocal cords diminishes the size of the lumen of the glottis and produces change in the quality of the sound, but it is not as marked as in morbid growths; and while the quality of the sound is changed, it lacks the harshness. Inflammatory thickening and exudations (œdema glottidis) produce the same changes as new growths. Croup (laryngeal diphtheria) and spasmodic conditions of the larynx produce a form of stridor, which, on account of its musical quality, may mask the vesicular element of the breath sounds so that the only sound heard over the surface of the chest will be more or less bronchial in type. It must be especially remembered that the introduction into the larynx of intubation tubes, or the presence of a tracheotomy tube, may cause the breath sound heard over the lung to be distinctly tubular. This frequently causes the diagnosis to be made of secondary pneumonia, and a corresponding gloomy prognosis in cases of diphtheria in young children.

It will be *diminished* by conditions which will hold apart the vocal cords.

Especial care must be taken in judging of the pulmonary condition by auscultatory signs when disease of the larynx, which causes a diminution of the glottic sound, is present, as the effect of changes in the laryngeal sound may be so marked as to prevent bronchial breathing being produced in well-marked consolidation of the lungs.

Influence of the Bronchi.

The condition of the bronchial tubes may modify the respiratory sounds in their normal elements, or add new sounds, as adventitious sounds of râles.

When the lumen of the tubes is narrowed, either by congestion, as in acute bronchitis, by growths within or by pressure from without, the intensity of the murmur over that portion of the lung which they supply with air may be increased or diminished, according as the amount and movement of tidal air is influenced. When the narrowing causes no diminution in the amount of air passing to the lung beyond, then, on account of the narrowing, the movement of the air through the narrowed portion will be more rapid and forcible. Inspiration will be harsher in character, while expiration will be more blowing, higher pitched and of longer duration.

If, on the other hand, changes in the bronchi interfere with the movement of air, so that it reaches the bronchioles reduced in amount and force, inspiration will be feeble, the vesicular element especially weak, and expiration perhaps inaudible.

Diminution in the intensity of the respiratory murmur is most marked when the smaller bronchi are involved. These changes in intensity may be bilateral or limited to one lung or a portion of one lung, according to the morbid processes.

The abnormal or adventitious sounds present in diseases of the bronchi are the dry and moist râles, according to the changes in the bronchi and nature of their contents.

Dry Râles.—When the lumen of a bronchus is so abruptly narrowed, either by contraction of the muscular fibres or by thick, tenacious secretion, as to produce conditions like those of a wind instrument, as the tidal air passes over the obstruction into the relatively wider lumen beyond, vibrations are set up and a sound produced as in the larynx at the glottis.

The character of the sound will depend upon the degree of narrowing, the size of the tube and the force of the current, and the adventitious sound will be added to or modify that made at the glottis.

When the narrowing is relatively slight, the sound produced will be of a harsh, blowing, non-musical character, similar to that made normally at the larynx. At the surface it will have a well-marked bronchial character, most marked with expiration, and almost identical with that heard when the lung is consolidated, from which it is differentiated by the signs noted by percussion and palpation. This sound may occur in bronchitis, asthma and pulmonary tuberculosis. In the latter disease it may be associated with more or less consolidation.

When the narrowing is more marked, the sound will have a more musical quality, being a type of stridor already described.

When the constriction is in a tube of the second or third division, it produces a deep-toned, low-pitched, musical sound (sonorous râle).

When in the smaller tubes, the sound is shriller, higher pitched, whistling (hissing or sibilant râle).

The division into sonorous and sibilant râles is an arbitrary one, and one type merges into another, according to the size of the tube and nature of the obstruction.

The dry râles are not permanent, being evanescent, appearing and disappearing; now being heard at one place, then over another area.

The effect of these râles on the normal respiratory murmur varies according to their number and intensity, and whether or not the constriction is so marked as to interfere with the movement of tidal air and prevent the production of the normal vesicular element.

The persistence of the vesicular sound is an important diagnostic sign, indicating the degree of obstruction. Students frequently fail to appreciate the vesicular murmur even when present, as the sibilant and sonorous sounds are so striking as to engross the attention.

The special significance of these râles being heard in inspiration or expiration, or limited to one or the other, will be considered under the diseases.

Moist Râles.—When the bronchi are more or less filled with fluid, there will be present the different varieties of moist râles that have already been described (page 115).

The conditions that produce dry and moist râles may affect the entire bronchial tract, as in general bronchitis, or may be limited to one lung or to a portion of a lobe. The wide or limited distribution of the adventitious sounds is of important diagnostic value.

The Effect of Plugging of a Bronchus. — Complete closure of a bronchus by compression from without, as by an enlarged gland or aneurism, by the plugging with secretions or plastic exudation, as in bronchitis and pneumonia, or by a growth within the tube, as in tuberculosis, produces marked diminution or complete absence of the breath sounds, according to the site of the obstruction. It influences the production of sound by the tidal air and the transmission by the tissues. Vibrations that are present in the column of air are arrested at the point of occlusion, and as there is no movement of air beyond that point, no sound is made at the junction of the alveoli with the infundibuli, and consequently the vesicular quality of the respiratory murmur is wanting.

Important changes occur in the area that is supplied by the occluded bronchus. The air beyond the obstruction is rapidly absorbed, and when the plug acts as a ball valve the air may be forced out during expiration, but not replaced during inspiration. Removal of air causes relaxation of the lung, and more or less complete atelectasis. As will be shown later, this loss of tension causes the tissue to be a poor conductor of vibrations, and the expiratory portion will be especially feeble or absent.

When a large bronchus is occluded, no sound will be heard over the portion supplied, so that even bronchial breathing may be absent in complete consolidation of the lung, as in croupous pneumonia. If a small branch only is involved, then feeble breath sounds may be heard over the affected area, and, the surrounding portion of the lung being overdistended, exaggerated or compensatory breathing is present. (Fig. 25.)

(B) CONDITIONS MODIFYING TISSUE VIBRATION AND THE CONDUCTION OF SOUND BY THE TISSUES.

The Influence of Tension.—The elasticity of the tissues of the respiratory tract and their degree of tension has an important effect not only on the intensity, but also on the quality and pitch of the breath sounds.

When the elasticity and tension are increased, the tissues are more easily influenced by the aerial vibrations present in the bronchi and air spaces. The vibrations are shorter; the tissues become better conductors of sound; the inspiratory sound is consequently more intense, has a sharper vesicular quality and the pitch will be slightly raised. The expiratory sound is louder, more intense, is longer, has a more marked blowing quality, often with a suspicion of a bronchial element. Age has an important effect on the elasticity and tension of the respiratory apparatus. In the child not only is the resiliency of the tissues greater, but up to twelve years of age the relatively more rapid development of the thorax than the lung causes the tension to be much greater than in the adult, and is one of the factors that causes the respiratory sounds in children to be distinctive enough to be called "puerile breathing" and stand out as a type.

Exaggerated or compensatory breathing is due to increased tension, as well as to changes in the volume and force of the tidal air.

With advancing years, and when pathological changes occur, as in emphysema, there is a loss of elasticity; and, although the lung may be distended beyond the normal, there is a lowered tension, and the respiratory sounds are feeble and lower pitched; inspiration is shorter, while expiration is prolonged.

Influence of the Increase of Tissue in the Walls of the Bronchi and Alveoli on the Respiratory Sounds.—When considering the production of normal vesicular murmur, it was stated that the thin, membranous structure of the walls of the bronchioles and alveoli was chiefly influenced by the vibrations of the air contained in them; also that this tissue was a poor conductor of the vibrations brought to it from above through the more solid tissues of the walls of the bronchi, and that these facts accounted for the absence of the bronchial or tubular sound in the normal respiratory murmur.

When the normally light membranous structure of the periphery of the lung is changed into denser connective tissue, as occurs in interstitial or fibroid changes in the interlobular septa (in interstitial pneumonia, fibroid phthisis and certain types of pulmonary tuberculosis), the new tissue, being more homogeneous with the walls of the larger bronchi, will conduct the bronchial sound from the deeper portion of the lung direct to the surface. The change may be so slight as only to give a slightly prolonged expiration. The ear may detect two types of breathing in varying proportions—the normal vesicular murmur and an added bronchial element, most marked in expiration. This mixed breathing is called broncho-vesicular. The proportion and character of the tubular quality will depend upon *(a)* the amount of new tissue; *(b)* the extent of its penetration into the lung and the size of the bronchial tubes reached.

Broncho-vesicular breathing varies from the faintest tinge of the bronchial sound to that in which the tubular sound predominates, and the vesicular element can only be faintly detected. (Fig. 25.)

When the air in the alveoli is replaced by coagulated exudate or tissue, as occurs in pneumonia, hemorrhagic infarction and tubercular infiltration, then during inspiration the vesicular quality is absent, because there is interference with the amount and force of tidal air, and the aerial vibrations are unable to modify the tissue vibrations present. The consolidated lung, being a good conductor, allows the bronchial sound present in the deeper portion of the lung to be heard.

When the consolidation is very slight, the sound from both the normal and consolidated lung is brought to the ear, so that the only change noted may be limited to a prolongation of expiration, with a slight raising of the pitch, *i. e.,* the faintest type of broncho-vesicular breathing.

If the consolidation reaches only to the small bronchi, then the breathing, while bronchial, is of a soft, blowing quality; the pitch is raised; the intensity corresponds to the pitch, and the expiratory sound is prolonged, more tubular and higher pitched than the inspiratory, and a short pause is noticed between inspiration and expiration.

When a medium-sized tube is involved, the quality will be more bronchial; the pitch will be higher; the intensity greater; the expiratory sound will be higher pitched and longer than the inspiratory, and will have a more markedly tubular character. This is usually described as bronchial breathing.

When an entire lobe is involved, then the sound heard is that conveyed from the large bronchi. It has all of the characteristics of that heard normally over the trachea, and is called pure bronchial, tubular or tracheal breathing.

The auscultatory signs heard at the surface in consolidation of the lung, whether due to fibrosis of the stroma or filling of the air spaces, are not always as typical as has just been described.

When the consolidated portion reaches the surface, and is immediately beneath the ear, then the breathing will be typical of the pathological changes, but when it is deeper and is covered by lung that is normal, or in a state of compensatory distension, the bronchial element may be entirely masked by the normal or exaggerated breath sound of the overlying portion of the lung, or it may be faintly detected, especially in expiration, where the first part has a fairly normal quality, but the latter part is prolonged, high pitched, more blowing and bronchial. (Fig. 25.)

This type of breathing, according to the intensity of the bronchial sound, as has been described as "broncho-vesicular breathing," "prolonged, high-pitched expiration" or one of the types of the so-called "metamorphosing breathing."

The influence of changes occurring in the pleura on the bronchial breathing of consolidation will be considered later.

Influence of the Increase in the Size of the Air-Containing Spaces (cavity formation and bronchiectasis).—The destruction of lung tissue and the formation of cavities, which are at least partly empty and connect freely with a bronchus, cause a modification of the bronchial breathing, which is called *cavernous* or *amphoric,* according to the dimensions of the cavity, the condition of the tissue forming its walls and the size of the bronchus opening into it.

Much confusion will be avoided if it be remembered that tubular, cavernous and amphoric are all types of bronchial breathing; that they vary in degree, and often approach each other in character. While they have a general resemblance, the differentiation is made by the quality and pitch of the sound.

The bronchial sound that is brought to the cavity is resonated, as when one blows over the mouth of a bottle. When the walls of the cavity are flaccid and of low tension, a peculiar hollow sound is produced, which is called *cavernous.* The inspiratory portion is soft, blowing, hollow, but less tubular, and lower pitched than the tracheal sound. The expiratory is more blowing, wavy or puffy; the pitch is *lower* than that of the inspiratory, which distinguishes it from bronchial breathing, in which the pitch of expiratory is higher than that of the inspiratory sound.

The sound heard over the trachea, behind, serves as a standard for bronchial breathing with which to compare doubtful cavernous breathing.

When the walls of a cavity are rigid and tense, as when composed of dense, fibrous tissue, when surrounded by consolidated lung, or when bound to the chest wall by firm pleuritic adhesions, the sound produced is *amphoric.*

It is a hollow, blowing sound, similar to the cavernous, but has a harsh, metallic or slight echo quality. The tubular element is more marked, but less than in bronchial breathing, and the expiratory sound has a lower pitch than that of inspiration.

The terms cavernous and amphoric, used to designate the breath sound, heard over cavities, are sometimes uncertain in their application, owing to the individual interpretation of the sound heard. This is due to the fact that the walls of cavities vary in tension. A type of breath sound is frequently heard which has the qualities of the cavernous and amphoric breathing in various degrees, and may be called caverno-amphoric or amphoro-cavernic, as either type predominates.

When the cavity is filled with fluid or the bronchus leading to it is occluded, cavernous or amphoric breathing may be absent, either temporarily or permanently.

When a bronchus opens freely into the pleural cavity filled with air, a peculiar, intense type of amphoric breathing is heard. This has been called the "lung fistula sound."

(C) CONDITIONS MODIFYING VOCAL RESONANCE.

The difference in the sounds heard over the chest when a person breathes, whispers or speaks depends upon the character of the vibrations made at the glottis.

Whispered Voice or Whispered Resonance.

In order to intensify the sound made at the larynx, the patient may be instructed to breathe noisily, or, what is better still, to whisper with different degrees of loudness. The whispered voice, in addition to being more intense than the breath sound, gives the articulate elements of speech—the consonants. The clearness with which they are heard over the different regions of the chest indicates the extent and degree of the changes that have occurred in the power of the lung to conduct sound to the surface.

The whispered voice is heard with expiration, and when patients are too weak to take deep breaths we can increase the intensity of the sound heard on expiration without exhausting the patient by having him whisper.

Normally, the whispered words are conveyed to the ear as soft, diffuse, indistinct sounds. Over those portions of the lung where tracheal, laryngeal and bronchial breathing normally exist, the whispered words are heard with greater distinctness, and the words will be recognized more or less completely.

Conduction of the whispered voice will be increased or diminished in the same manner and under like conditions as the breath sounds. It is especially valuable to determine the presence of a slight amount of consolidation, when the whispered words will be brought to the ear with increased distinctness.

As consolidation increases in extent and involves the larger tubes, the whispered words become more and more distinct, until it is possible not only to recognize the whispered words or numbers (whisper-bronchophony), but also to detect the articulate parts of speech, as the consonants (whisper-pectoriloquy).

The Spoken Voice or Vocal Resonance.

The vibrations of the spoken voice are so powerful as to influence the entire amount of air within the bronchi and alveoli, so that they are not only heard at the periphery as normal resonance, but are also felt as fremitus. The open bronchi, with their tense, elastic walls, act as speaking tubes, and the sound made at the glottis is modified in its conduction to the surface, as follows: (1) By reflection, reverberation or echo within the tubes, so that confusion of the sound occurs. (2) By diffusion in the ramifications of the bronchi and alveoli, so that it is weakened or less intense.

In the larynx and trachea little change occurs in the sound, except that the consonants are not heard as distinctly, but the intensity of the sound seems to be slightly greater than at the mouth.

In the larger bronchi, as heard over the sternum and parasternal portion of the infraclavicular space in front, and upper portion of the interscapular space behind, the sound is more indistinct, the words are blurred, but still distinguishable, and the pitch of the sound is slightly higher than in the trachea.

This sound is called "normal bronchophony," and may be heard over other regions of the chest when consolidation of the lung tissue transmits it unmodified to the surface.

With each division of the bronchi the sound is still further reflected, confused and weakened, as were the breath sounds, until at the surface of the lung the articulate parts of speech are absent; the words are not distinguishable, and it becomes an indistinct humming, buzzing sound, *normal vocal resonance.*

Normal vocal resonance will vary in individuals, according to the character of the voice, as has been mentioned under "Palpation."

It will be influenced by the same changes in the bronchi and alveoli that modify breath sounds and the fremitus of palpation.

Vocal resonance may be absent, feeble or exaggerated. Just as in proportion to the pulmonary consolidation, the expiratory part of the respiratory murmur varies in intensity, pitch and quality, from the faintest suspicion of bronchial sound up to pure bronchial breathing, so vocal resonance varies in the clearness with which the articulate sounds are heard from a slight increase of normal resonance up to bronchophony, where the words are heard more or less distinctly, but confused and associated with vibrations of the chest wall.

PECTORILOQUY, as its name implies, sounds as if the patient was speaking directly through the chest, or as if we were listening directly over the larynx through a solid medium, as a block of wood. It is an exaggerated form of bronchophony. Its characteristic feature is the remarkable distinctness with which the consonants of the spoken words are heard. It is best detected with the whispered voice, as there is less confusion from reverberation.

Pectoriloquy is heard over consolidated lung that is in a high state of tension, and that extends to the large bronchi.

Over pulmonary cavities two kinds of spoken and whispered resonance are heard: *(a)* cavernous and *(b)* amphoric.

CAVERNOUS RESONANCE is produced in cavities with thin, lax walls. It is a type of bronchophony. The sound is hollow, low pitched, and has a puffing, blowing character. With the spoken voice, the words are confused, having a slight echo quality. With the whispered voice, the words are more distinct, but not articulate.

AMPHORIC RESONANCE occurs in cavities with tense walls, and is usually associated with amphoric breathing. The sound is a type of pectoriloquy, in that it is distinctly articulate, with a hollow, ringing, well-marked echo quality and high pitch.

ÆGOPHONY is a form of vocal resonance that occurs in certain stages of effusion in the pleural sac. The voice is not as intense as in bronchophony, but is high pitched, tremulous, with a marked nasal sound, so that it has been compared to the bleating of the goat; but it is more like the voice of a person with a cleft palate. It is not associated with increased vocal fremitus. The condition of the lung allowing its production will be considered under "Pleurisy with Effusion."

Auscultation of the Cough.

The sound produced by coughing is frequently utilized in diagnosis, but it is not as reliable as vocal resonance. In the normal lung it is heard at the surface as an indistinct, sharp sound, accompanied by sudden movement of the chest wall, due to the action of the expiratory muscles. In varying degrees of pulmonary consolidation and in cavities it has been named bronchophonic, cavernous and amphoric cough, having the same features as vocal resonance under similar conditions.

On account of the forcible expulsive power of cough, a larger amount of air than usual is driven out of the lung, and the succeeding inspiration is correspondingly increased in volume. This allows slight changes in the bronchi and pulmonary tissue to be detected by the character of the vesicular murmur. "A post-tussive suction sound" or "india rubber-ball sound" is frequently heard with the inspiration immediately after the cough when cavities with soft, yielding walls are present. It is a sucking, semi-sonorous, low-pitched sound, usually accompanied by a few mucous clicks or medium-sized moist râles. It is a very important and reliable sign of cavity formation.

Coughing has a marked effect on moist râles. When the fluid contents of the tube are dislodged, it may cause them to disappear entirely, or to change small râles to those of larger size. When the secretion is in small cavities, with more or less occlusion of the bronchial tubes leading to them, no râles may be detected during quiet, or even forcible, breathing, but are heard after coughing.

THE BELL SOUND, BELL TYMPANY, OR COIN RESONANCE.—In pneumothorax, or when a large cavity is present in the lung close to the surface, if percussion is made on the anterior portion of the chest with two coins while the auscultator listens behind, there is heard over the affected area a distinct ringing bell- or anvil-like sound. In order that this air-containing space may act as a resonator, it is necessary that it be of a certain size, with tense walls.

VEILED PUFF.—This is a short, high-pitched, puffing sound, which is added to the inspiratory murmur toward the end of inspiration. It has been considered diagnostic of small sacculated bronchiectatic cavities.

(D) INFLUENCES OF CHANGES IN THE PLEURA ON THE BREATH SOUNDS.

The normal pleura, being very thin and almost homogeneous with the tissue of the walls of the alveoli, does not affect the transmission of the respiratory sounds. When the pleura is thinly covered with a sticky secretion, different varieties of friction sounds are heard, which may mask the pulmonary sound.

When the pleural changes cause pain on breathing, movement of the affected side is restricted, and there is a corresponding decrease in the intensity of the vesicular murmur. Generally all increase in the thickness of the pleura, whether from plastic exudation or interstitial changes, interferes with the transmission of vibrations to the surface, and influences the intensity of the breath sounds.

"The motion of sound, like all other motion, is enfeebled by its transmission from a light body to a heavy one" (Tyndall), so that the vibrations in the thin membranous walls of the alveoli are transformed into vibrations in the thickened pleura, with loss in their intensity which is proportionate to the change in the homogeneity or density of the pleura.

Changes in the pleura, especially the pulmonary portion, which render it less elastic or distensible, curtail the normal inspiratory enlargement of the lung, and thus decrease the amount and force of tidal air and enfeeble the breath sounds. This effect is especially marked over the apices and lower borders of the lungs.

Collections of fluid, air or the formation of new growths in the pleural sac separate more or less widely the two surfaces of the pleura, and allow the lungs to retract. This produces loss of tension in the pulmonary tissue and weakens the vibrations at the surface of the lung, so that the breath sounds are heard very feebly or not at all.

The occurrence of bronchial breathing and ægophony at the upper level of the fluid effusions will be explained under "Pleurisy with Effusion."

Adhesions between the two surfaces of the pleura may cause marked decrease in the breath sounds by preventing normal movements and limiting expansion.

When pleural adhesions keep the pulmonary tissue tense, or are associated with increase of pulmonary tissue, as occurs in interstitial pneumonia and the fibroid type of phthisis, they may form homogeneous bands of connection between the ribs and the bronchi, and so permit the breath sounds to be heard where ordinarily they are absent.

(E) INFLUENCE OF THE THORACIC WALL ON BREATH SOUNDS.

The bony thorax acts as a sounding board for the vibrations conveyed to it, and influences the intensity of the breath sounds according to its elasticity. At the same time, by its rigidity, it affects the movement of the ribs and modifies the amount of tidal air and the intensity and duration of the vesicular murmur.

The inspiratory portion of respiration being a muscular act, its intensity, duration and rhythm are easily modified by changes affecting the muscles.

The rhythm may become irregular, jerky or interrupted through the imperfect or irregular contraction of the ordinary muscles of inspiration. It may be due to nervousness of the patient, disease of the nervous system or of the muscles themselves, or to pain either in the chest wall or in the pleura.

Expiratory movement, although normally a passive act, may be altered by the muscles of expiration in the same manner as inspiration.

The thickness of the soft parts will also affect the clearness with which the respiratory sounds are heard, in a manner similar to its effect on vocal fremitus. (See "Palpation.")

CHAPTER VII.

DIAGNOSIS OF DISEASES OF RESPIRATORY TRACT.

BRONCHITIS.

Bronchitis is an inflammation of the different tissues of the bronchial tubes. It may be acute or chronic. It may involve a limited portion only, as the large tubes, or the entire bronchial tract. Primary acute bronchitis usually affects the bronchi of both lungs to a nearly equal extent. Accompanying or pre-existent disease of the lung causes the bronchitis to be limited to a portion of one lung only, or to be much more intense on one side than on the other.

Acute Bronchitis.

The physical signs present in acute bronchitis depend on: (1) The size of the bronchial tubes involved. (2) The nature and extent of the changes in the mucous membrane. (3) The character, amount and distribution of the secretion or exudation present in the bronchi. (4) The influence of changes in the mucous membrane, and the presence of secretion on the movement of air in the bronchi.

As the lumen bronchial tube has a most important bearing on the intensity of the physical signs and the gravity of the disease, it furnishes a basis for classification of the varieties of bronchitis. Acute bronchitis may be divided into:

A. Bronchitis of the Larger Tubes. — This includes involvement of the trachea and the larger cartilaginous tubes, whose diameter is so great that the tumefaction of the mucous membrane and the presence of more or less fluid secretion or inflammatory exudation does not cause marked obstruction or interference with the movement of air.

B. Bronchitis of the Middle-Sized Tubes.—An inflammation of the medium-sized and smaller cartilaginous tubes, whose lumen is narrowed to a greater or less extent, with a more or less interference with the movement of air and a corresponding production of physical signs. *Synonyms.*—Simple bronchitis, acute bronchial catarrh.

C. Bronchitis of the Smaller Tubes.—An inflammation of the smallest cartilaginous and non-cartilaginous tubes down to the lobular bronchioles, and characterized by marked obstruction of the bronchi and imperfect ventilation of the lung. *Synonyms.*—Acute capillary bronchitis, acute suffocative catarrh.

D. Bronchitis of the Smallest Tubes, including lobular bronchi, and extending into the intralobular bronchioles and air-passages, with tendency to lobular consolidation. This is characterized by extreme interference with passage of tidal air to and from the alveoli, with imperfect oxygenation of the blood. *Synonyms.*—Capillary bronchitis of infancy and old age; capillary bronchitis with broncho-pneumonia; peripneumonia nothia.

PHYSICAL SIGNS.—As the classification of bronchitis given above is largely arbitrary, and as we rarely find the disease limited to the different divisions of the bronchi, so also the physical signs that are present in one variety of bronchitis merge more or less into those of the other varieties. It is best to compare the physical signs of the different varieties.

INSPECTION.—*A. Bronchitis of the Larger Tubes.*—As there is neither change in the pulmonary tissue nor interference with the movement of air through the bronchi, there is no alteration in the shape and size of the thorax. The respiratory movements may be slightly increased, both in frequency and depth, but the two sides move symmetrically, and expansion is full and free.

B. Bronchitis of the Middle-sized Tubes.—In mild cases no change is noted in the size or form of the thorax, and the respiratory movements are but slightly increased in force and frequency. In severe cases, in proportion to the narrowing of the lumen of the bronchi by inflammatory swelling or secretion and interference with the free movement of air, the respiratory movements are more rapid and labored, expansion being restricted when tubes of smaller calibre become involved and the above signs become increased in intensity.

C. Bronchitis of Smaller Tubes.—There is a marked movement of the upper portion of the chest, with violent action of the accessory inspiratory muscles; the expansion of the chest is chiefly confined to upper portion; there is loss of expansion in lower portions of the chest, and diaphragmatic action is restricted. As the case becomes more severe, and the interference with the movement of air through the small bronchi becomes more marked, two types of dyspnœa may develop.

(1) Inspiratory Dyspnœa. In this form the greatest interference is with air passing into the alveoli; the action of the accessory muscles of inspiration is especially great, causing marked elevation of the upper portion of the chest, while the partial vacuum that is produced in the lower portion of the lung causes depression of the intercostal spaces, loss of expansion of the lower portion of the thorax, and, in infants and young children, yielding of the chest walls, depression of the lower ribs and retraction of the abdomen.

(2) Expiratory Dyspnœa. In this form entrance of air into the alveoli is fairly free, and the chief difficulty is with the tidal air passing out of the alveoli, due to plugging of the small bronchi with secretion or to spasm of the bronchi themselves. This gradual overfilling of the alveoli (acute emphysema) causes enlarge-

ment of the thorax; the movements of inspiration are short and followed by a pause, while those of expiration are prolonged and labored. During expiration the accessory muscles are brought into play, and there is bulging of the intercostal spaces and supraclavicular fossa.

D. Bronchitis Involving the Lobular Bronchioles and Alveoli.—The cyanosis is more marked; the respirations are rapid; from thirty to forty, and may reach sixty, or even more, per minute. The dyspnœa is chiefly inspiratory, with deep depression of the supraclavicular fossa and intercostal spaces, and, as the bases of the lungs are chiefly involved in children, there is marked drawing in of the lower ribs, ensiform cartilage and epigastrium.

PALPATION.—*A. Bronchitis of Large Tubes.*—Surface temperature slightly raised; respiratory movements normal (free, rhythmical and symmetrical); vocal fremitus normal, except when larynx is also involved, when it is reduced in direct ratio to the huskiness of the voice.

B. Bronchitis of Middle-sized Tubes.—In mild cases the respiratory movements and vocal fremitus are within range of normal. In severe cases, with marked congestion and tumefaction of the mucous membrane and thick, tenacious secretion, the movements are slightly more rapid and somewhat labored, vocal resonance is diminished, and when large bronchi are plugged, there is restricted motion over portion of lungs supplied by occluded tubes and absence of vocal fremitus. Rhonchal fremitus is sometimes present.

C. Bronchitis of Smaller Tubes.—In mild cases vocal fremitus may be normal or slightly diminished. In severe cases, in proportion to the obstruction, the movements will be rapid and labored, and vocal fremitus feeble or even absent. In children, and sometimes in adults with thin, elastic chest walls, rhonchal fremitus is present.

D. Bronchitis Involving Smallest Bronchioles and

Alveoli.—Vocal fremitus is diminished, or even absent, when obstruction is extreme. When inflammation has extended to the alveoli, causing consolidations of lung (broncho-pneumonia) close to the costal surface, vocal fremitus may be increased.

PERCUSSION.—*A. Bronchitis of the Large Tubes.*—As there is no change in the structure or tension of the pulmonary tissue or size of the air-containing spaces, percussion sound will be normal in quality, intensity, pitch and duration.

B. Bronchitis of the Middle-sized Tubes.—Percussion sound is normal unless one of the tubes is plugged. If the portion of lung supplied by occluded bronchus is in contact with costal surface, the percussion sound will have a slight tympanitic quality. If the surrounding portion of the lung that is in state of compensatory emphysema is reached by percussion, the sound will be hyper-resonant.

C. Bronchitis of Smaller Tubes.—In mild cases slight dullness in percussion may be present over lower portion of lung. In severe cases over portion of chest which is in a state of more or less compensatory emphysema, there is a varying degree of hyper-resonance, while over the lower portion, when there is inspiratory dyspnœa, imperfect expansion, collection of secretion in bronchioles and alveoli, with partial collapse of the lung; there is dullness, with slight tampanitic quality. When the dyspnœa is expiratory, with distension of the alveoli, there is hyper-resonance.

D. Bronchitis Involving Bronchioles and Alveoli.—Over areas of secondary bronchial pneumonia, if near the surface and of sufficient size, the percussion is dull in proportion to the extent of consolidation. As surrounding vesicles are frequently in a state of collapse, the note may be dull, with tympanitic quality. When the consolidated area is small and overlapped by distended emphysematous lobules, the percussion note may be normal or hyper-resonant.

AUSCULTATION.—*A. Bronchitis of Large Tubes.*—In mild cases there is no change in normal breath sounds. In the more severe types of the disease, during the dry stage, the normal tubular sound heard over the trachea and large bronchi has a harsher quality, due to the slight narrowing of the tubes. When secretion is present, if tenacious, it may produce local narrowing, causing coarse, sonorous râles. If the secretion is more liquid, a few large moist or mucous râles, which are easily removed by coughing, may be present. In the adult marked auscultatory signs are the exception; in children they are present more frequently. Vocal resonance is unchanged.

B. Bronchitis of Medium-sized Tubes.—In mild cases the breath sounds over the lung are normal, except that during the dry stage the quality is slightly harsher and the expiratory part of the sound is longer, but unchanged in quality and pitch. When the amount of fluid present in the tubes is fairly abundant, large and medium-size liquid, bubbling râles are heard during both inspiration and expiration. In more severe cases, when the lumen of the bronchi is narrowed by the congested and swollen mucous membrane during the dry stage, the respiratory murmur is less intense than normal during inspiration, while in expiration it is prolonged and has a faintly sonorous quality, which may be mistaken for a type of bronchial breathing. From this it is differentiated by being heard over both lungs and by the absence of the associated signs of consolidation, dullness and increased vocal fremitus. When the fluid is abundant and tenacious, sonorous and sibilant râles and rhonchi are present. These adventitious sounds may mask the respiratory murmur; they are apt to be inconstant, are easily changed in character by coughing and deep breathing, and are heard over both lungs with equal intensity. When the secretion is more liquid, dry râles are less prominent, and heard chiefly over the upper portion of the lung, while large and medium-sized liquid, bubbling râles predominate. They are most abundant at the base, as the secretion tends to gravitate to the most dependent portion of the lung, especially in children and feeble adults. As the smaller tubes become involved, either by extension of the inflammation or by gravitation of the secretions into them, the dry râles become more sibilant and the moist râles finer and more crepitating. Vocal resonance is normal or slightly diminished.

C. Bronchitis of the Smaller Tubes.—The respiratory murmur may show very little change during the dry stage; the intensity of inspiratory sound is diminished, the quality being somewhat harsher and dry, the pitch unchanged. The expiratory sound is more intense than that of inspiration; the sound is prolonged, and may have a harsher, blowing quality. When liquid is present, sibilant râles and smaller mucous râles accompany both sounds. In direct proportion to the severity of the case, the respiratory sounds are diminished in intensity, and may be obscured and replaced by hissing, sibilant râles and small liquid râles and crepitations. As the secretion accumulates in the bronchi, it tends to gravitate to the lower part of the lung, so that over this portion the small, bubbling and crepitating râles will be most marked, while over the upper portion of the lung the moist râles will be coarser and the sibilant râles more abundant.

D. Bronchitis Involving the Lobular Bronchioles and Alveoli.—Extension of inflammation into the lobular bronchioles and alveoli (broncho-pneumonia) is shown by the more or less intense bronchial quality of the breath sounds, especially marked during expiration; the râles heard over the areas of bronchial breathing are finer, more ringing or crepitating than those heard in simple bronchitis, and are most abundant just at the end of inspiration. Vocal resonance is increased over areas of consolidation.

Differential Diagnosis.—The diagnostic features of the different varieties of acute bronchitis are: (1) There is a bilateral distribution of all the physical signs. (2) In proportion to the severity of the disease, the smaller bronchi become involved, giving more marked physical signs over the lower portion of the chest. (3) The physical signs detected are those due to changes in the bronchial tubes only, *i. e.,* the presence of râles of varying size and interference with the movement of tidal air.

The diseases with which acute bronchitis is most liable to be confounded are:

Broncho-Pneumonia (Acute Catarrhal Pneumonia).—In both diseases the signs are bilateral, most intense at the base of the lung, and there are numerous râles. But in broncho-pneumonia there is not the same uniformity of distribution. Broncho-pneumonia gives, over the area of consolidation, increased vocal fremitus. Uncomplicated acute bronchitis, diminished vocal fremitus. Broncho-pneumonia, dullness of varying degree, according to amount of lung involved. Bronchitis, but slight change in resonance. Broncho-pneumonia, bronchial or tubular quality of the breath sounds, which is absent in bronchitis.

Acute Diffuse Pulmonary Tuberculosis.—The physical signs in this disease are bilateral, and at the beginning are almost identical with those of capillary bronchitis with small areas of broncho-pneumonia, but the general constitutional symptoms are graver. As the disease advances, the physical signs become more distinctive.

INSPECTION.—Tuberculosis: Respirations are more rapid; cyanosis out of proportion to dyspnœa; restricted motion over the entire chest. Retraction of the thorax most marked over upper portion of lung, and more marked at one apex than the other. Bronchitis: Retraction most marked over the lower lobes; generally increased fullness of upper portion of the chest in proportion to the retraction at base.

PALPATION.—Tuberculosis: Vocal fremitus may at first be diminished; later increased with extension of consolidation. Simple bronchitis: Vocal fremitus ne[illegible] slightly diminished.

Percussion.—Tuberculosis: Resonance early impaired, with slight dullness, due to change in the alveoli, and early pleuritic involvement. Dullness is most marked over the upper portion of the chest; there is increased sense of resistance over the part involved. Bronchitis: Percussion normal or hyper-resonant.

Auscultation.—The differential diagnosis rests chiefly on the course of the disease and the distribution of the râles. While in acute bronchitis, with the dyspnœa and cyanosis, there is a corresponding number of dry and moist râles, most marked at the base, in tuberculosis the number of moist râles is comparatively few, and they are as numerous, or even more so, at the apex as at the base; also there is generally an uneven distribution of the râles on the two sides. Later there is a tendency for the signs of consolidation to become more marked over different areas of the lung, and to be followed by signs of diffuse softening and cavity formation. Frequently it is possible to find in some portion of the lung, especially at the apex, an old focus, with its characteristic physical signs of long-standing consolidation.

Pulmonary Œdema.—The differential diagnosis of bronchitis from pulmonary œdema rests chiefly on the order of occurrence of moist râles. In acute bronchitis the larger tubes are primarily involved, and the râles first detected are those of large size. With the extension of the disease into smaller bronchi, râles of corresponding size are also present, and are heard over the entire lung. In pulmonary œdema fluid collects in the alveoli, and fine crepitating râles are first heard over the lower borders and base of the lung. The larger râles are only present when the œdema is extensive.

Chronic Bronchitis.

The physical signs of chronic bronchitis, when only the mucous membrane is involved, are similar to those of the acute variety. Long continuance of the cough is a bilateral distribution of all the physical signs. (2) In proportion to the severity of the disease, the smaller bronchi become involved, giving more marked physical signs over the lower portion of the chest. (3) The physical signs detected are those due to changes in the bronchial tubes only, *i. e.,* the presence of râles of varying size and interference with the movement of tidal air.

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INSPECTION.—Tuberculosis: Respirations are more rapid; cyanosis out of proportion to dyspnœa; restricted motion over the entire chest. Retraction of the thorax most marked over upper portion of lung, and more marked at one apex than the other. Bronchitis: Retraction most marked over the lower lobes; generally increased fullness of upper portion of the chest in proportion to the retraction at base.

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Chronic Bronchitis.

The physical signs of chronic bronchitis, when only the mucous membrane is involved, are similar to those of the acute variety. Long continuance of the cough interferes with the function of the lung, and repeated attacks of acute or subacute bronchitis develop secondary changes in the bronchi, alveoli and pulmonary tissue. These changes determine the different types of chronic bronchitis and cause its symptoms and physical signs to be more or less characteristic of the secondary changes, *(a)* bronchiectasis, *(b)* asthma or spasmodic bronchitis, *(c)* emphysema, *(d)* fibroid or interstitial pneumonia.

Types or Varieties of Chronic Bronchitis.—A. Simple chronic catarrh of the large tubes (winter cough); characterized by cough and slight expectoration, with tendency to intercurrent attacks of acute bronchitis. The structures of the lung are but slightly changed from the normal.

B. Dry catarrh; characterized by very little secretion from the mucous membrane, by constant or persistent cough, scanty or difficult expectoration, presence of dyspnœa. The constant and violent attacks of coughing early induce emphysematous change, and acute attacks of bronchitis may cause it to assume temporarily the physical signs of the spasmodic type. The physical signs are those of acute bronchitis during the dry stage.

C. Bronchitis with abundant secretion. Under this may be included (1) bronchorrhœa, of which two types are recognized, *(a)* cases in which the secretion is thin, watery, slightly tenacious, resembling the white of an unboiled egg. Severe paroxysmal attacks of coughing and dyspnœa occur in the morning, and may last for one or two hours, after which the chest is clear; or they may be repeated at longer or shorter intervals, followed by complete relief. From the character of the symptoms and expectoration, the names "mucoid asthma" and "bronchorrhœa serosa" have been given. *(b)* Cases with sero-purulent or muco-purulent expectoration, attended with almost constant coughing and frequent attacks of paroxysmal dyspnœa. These attacks rapidly induce structural changes in the lung of the emphysematous, bronchiectatic and fibroid types. (2) Fetid bronchitis. In this the secretions have undergone decomposition, and may occur in bronchi that have not been dilated, but is most frequently dependent upon dilatation of the bronchi and long retention of secretion. (3) Plastic bronchitis, in which the secretion is coagulated in the bronchi, occluding them by fibrous plugs. A still further subdivision of the types of chronic bronchitis has been made upon the secondary changes in the lung, as: D. Bronchitis with spasm of the bronchi, spasmodic or asthmatic bronchitis. E. Bronchitis with distension of the alveoli, emphysematous bronchitis. F. Bronchitis with marked fibroid or interstitial changes.

Physical Signs. INSPECTION.—A. There is no change in the size, shape or movements of the chest. B. Thorax early assumes emphysematous type. Movements at first are exaggerated, and are more frequent. Later, expansion interfered with. Expiratory dyspnœa. C. In simple serous bronchorrhœa the respiratory movements are similar to those of acute bronchitis; in chronic purulent catarrh and simple putrid bronchitis, in the early stages, there is no change in the size or movements of the thorax. Later it assumes the emphysematous or fibroid type. Plastic bronchitis: extreme dyspnœa. Loss of expansion on inspiration especially marked over base of lung when localized to small portion of bronchial tract or limited motion over affected area. D. Thorax gradually assumes more or less the condition found in emphysema; the movements of the chest are similar to those present during the attack of spasmodic asthma. E. Chest emphysematous in all respects. F. General retraction of the chest; loss of expansion.

PALPATION.—A. Negative. B. Negative or vocal fremitus may be slightly diminished. C. Bronchorrhœa; feeble vocal fremitus; rhonchi felt during acute paroxysmal attack. Plastic bronchitis; loss of vocal fremitus over occluded tubes. D. Vocal fremitus feeble; rhonchi frequently present. E. Vocal fremitus feeble or absent. F. Increase of vocal fremitus especially marked over regions of large bronchi.

PERCUSSION.—Percussion note is unchanged in the milder types. When the lung has undergone emphysematous changes the note is hyper-resonant. Fibroid and cirrhotic changes give more or less dullness, with woodeny quality.

AUSCULTATION.—A. Signs indefinite. Few large moist râles may be detected; especially if secretion is present. Over the large bronchi, the breath sounds may be slightly roughened in character. B. Dry catarrh; respiratory murmur may be unchanged. C. In (1) bronchorrhœa during the attack, large and small liquid râles and sibilant and sonorous râles may be present. Breath sounds obscured by loud noise of râles. After expulsion of mucus, the respiratory murmur may be normal. (2) In fetid bronchitis the râles are more or less constant. Breath sounds are feeble. (3) In plastic bronchitis over that portion of the lung supplied by occluded bronchus, there will be absence of all breath sounds. D. In spasmodic bronchitis, as the name implies, associated with the moist râles are the breath sounds and dry râles that characterize an acute asthmatic attack. E. In emphysematous bronchitis the breath sounds are feeble, especially the inspiratory sounds, the expiratory portion being prolonged, low pitched and usually attended with râles. F. In chronic bronchitis with fibroid change the inspiratory sound is feeble, but the expiratory portion is prolonged, high pitched and has a faint tubular character.

Differential Diagnosis.—Chronic bronchitis is separated from the acute forms by the history of the attack and the presence of secondary changes in the lung. The various types of chronic bronchitis may be associated with pulmonary tuberculosis.

BRONCHIECTASIS.

Bronchiectasis is a distinct dilatation of the bronchial tubes. It may be limited to a portion of one lobe only, or may involve to a greater or less extent the bronchi of one or both lungs. Bronchiectasis is never a primary disease, but is secondary to some pathological change in the walls of the bronchi which alters or destroys their elasticity. The dilating force may be (a) increased pressure within the tube caused by coughing; (b) traction on the walls, causing permanent enlargement of their calibre. This traction may be due to contraction of bands of fibrous tissue in the interlobular septa, especially when they are stretched from the bronchi outward to the pleura. The dilatation of the bronchi may be sacculated or cylindrical.

The diseases with which bronchiectasis may be associated as a complication are broncho-pneumonia, capillary bronchitis with atalectasis, chronic bronchitis affecting the large and medium-sized tubes, emphysema, obstruction of the tubes from pressure from without, interstitial change in the lung, as occurs in chronic interstitial pneumonia (cirrhosis of the lung), in fibroid phthisis, and in pleurisy with retraction.

Physical Signs.—The physical signs of bronchiectasis depend (1) upon the disease with which it is associated; (2) the nature of the dilatation, whether cylindrical or fusiform; (3) whether the air passes through the dilatation to structures beyond; (4) the localization and distribution of the dilatations;[1] (5) the proximity of the dilatation to the surface of the chest; (6) the size of the tubes involved; (7) the condition of the cavity, whether full or empty.

INSPECTION.—The chest conformation of bronchiectasis is not typical. The shape and size of the thorax are determined by that of the disease to which it is secondary or with which it is associated. Three types occur—the emphysematous, the retracted and the combination of both, where retraction in one portion of the chest is compensated for by emphysematous expansion, at other portions. In the emphysematous type the upper portion of the chest, especially the supra- and infraclavicular spaces, are markedly distended, while the lower portion shows more or less retraction. Motion over the affected area is restricted according to the type of the disease and the condition of the surrounding lung. In mild cases, and when the disease is localized, motion may be but slightly impaired. When general or due to obstruction of the bronchi, the affected side will be more or less retracted.

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[1] One lung, 52 per cent.; both lungs, 48 per cent. Of the unilateral cases the upper lobe was involved in 21 per cent.; middle lobe, 3 per cent.; lower lobe, 32 per cent., and the entire lung, 42 per cent. (FOWLER.)

Palpation.—Vocal fremitus varies with the condition of the lung surrounding the bronchiectatic dilatation. When involvement of the bronchi is associated with emphysematous dilatation of the surrounding tissue, vocal fremitus is normal or diminished. Vocal fremitus is absent in occlusion of the bronchi or filling of the cavities with secretion, and also when marked pleural thickening has occurred. It is increased when the lung is fibrous and the bronchial tubes are patent beyond the dilatation.

Percussion.—Percussion note varies with the extent of the lesion, the condition of the surrounding lung, the nearness of the dilatation to the surface and the quantity of secretion in the cavities. As many of these factors are subject to frequent change, variation in the degree of resonance is an important diagnostic sign. When dilatation of the bronchi is slight, and it is surrounded by normal lung, the percussion sound may be normal. When marked emphysematous dilatation with relaxation of the lung occurs, the sound will be hyper-resonant, with a somewhat tympanitic quality added, due to the dilatation of the bronchi. When the dilatation of the bronchus is large, and near enough to the surface to be influenced by the percussion, the sound will be tympanitic or amphoric, according to the size. There will be change in the note according as the mouth is open or shut. When dilatation is surrounded by indurated lung, the note is dull, high pitched, with peculiar tympanitic "boxy" or "boardy" quality. One of the distinctive features of the percussion sounds of bronchiectasis is the localization of the change to the middle or lower third of the chest, especially over the back.

AUSCULTATION.—The most important sign is the blowing, hollow, but not tubular, sound when the dilatation is cylindrical, and a cavernous sound when the dilatation is sacculated. Both the inspiratory and expiratory breath sounds are interrupted and wavy, the blowing or cavernous quality being most marked just at the end of inspiration (veiled puff). Moist râles are present according to the amount and distribution of secretion in the bronchi and cavity. When the dilatations are cylindrical, the râles will have the characteristics of those heard in simple bronchitis of large and medium-sized tubes. *But the size of the râles is larger than would be found if the tubes in that region were of normal calibre.* In sacculated dilation the râles are larger, more gurgling, with an occasional croaking, sonorous râle. When the dilatation is surrounded by fibrous lung, the râles have a ringing or metallic quality. The occurrence of acute general bronchitis will change the character of the physical signs present over the cavity according as the bronchi are narrowed by inflammatory swelling or cavity filled with liquid secretion. With the subsidence of acute bronchitis, instead of the breath sounds returning to normal, the characteristic sounds of dilatation are heard. Vocal resonance over the cavity and whispered cavernous or amphoric breath sounds may be heard, according to the size and condition of the cavity and the surrounding lung.

Differential Diagnosis.—Bronchiectasis must be differentiated from other diseases of the lung in which cavity formation occurs. The character of the physical signs heard over the site of dilatation in bronchiectasis is not pathognomonic, and a diagnosis of the nature of the cavity can only be reached by exclusion.

Pulmonary Tuberculosis.—Pulmonary tuberculosis is the most frequent cause of the cavities found in the lung.

LOCATION.—Non-tubercular, solitary bronchiectatic cavities rarely involve the apices. It is most common at the base, but may involve the middle or lower third of the lung. Tuberculosis is especially likely to involve one or both apices primarily, extending to the middle and lower portions. In bronchiectasis the changes in the portion of the lung involved are more or less uniform. In phthisis limited portions of the lung may show the different stages of the diseases.

INSPECTION.—In bronchiectasis the retraction is most marked over the lower portion of chest; the upper portion may be normal or show in supra- and infraclavicular spaces emphysematous dilatation. In tuberculosis the retraction is most marked in the upper portion of the lung; the supra- and infraclavicular spaces are depressed. In bronchiectasis the patient is fairly well nourished, and there is more or less duskiness of the skin, congestion of the mucous membrane, fullness about the lips and nose and the jugulars are distended. The tuberculous patient is emaciated, the mucous membrane and skin pale and anæmic and there is no distension of jugulars. In bronchiectasis the heart is displaced horizontally towards the diseased side; epigastric pulsations are present. In tuberculosis the heart is displaced upwards and obliquely, and without epigastric pulsation except when it involves the base of the lung secondary to changes in the pleura.

PALPATION.—In bronchiectasis vocal fremitus may be diminished, except when marked fibroid induration is the cause of the dilated bronchi. In tuberculosis vocal fremitus is generally increased in proportion to the amount of consolidated lung tissue around the cavity.

Percussion.—Bronchiectasis: The supraclavicular region is rarely dull; its note is generally hyper-resonant or emphysematous. Dullness most frequently present over lower portion of chest. Tuberculosis: Dullness at the apex, becoming more intense as consolidation advances. Cavernous, amphoric or cracked-pot percussion always follows a first stage of dullness.

Auscultation.—Bronchiectasis: The blowing, hollow sound of cylindrical dilatation and the cavernous tubular sound of the sacculated form are unattended by any signs of pulmonary consolidation. Normal breathing sounds may be heard between the areas of cavernous breathing. In tuberculosis the sound is cavernous or amphoric. Bronchial breathing is heard over surrounding lung. In bronchiectasis the signs indicate that the cavities are of uniform size. In phthisis the cavities are rarely uniform. In bronchiectasis the size of the cavities remains stationary. In phthisis the cavities gradually enlarge, with associated signs of softening.

General Bronchitis and Fetid Bronchorrhœa.—Bronchiectasis is distinguished from general bronchitis by absence of fetor; from simple fetid bronchitis or bronchorrhœa the differential diagnosis is often extremely difficult.

The following signs are most important: Hollow, blowing or cavernous or amphoric breath sounds, according to the type of cavity. In general bronchitis vesicular murmur is present, but diminished or feeble; cavernous breathing not present. Size of râles has an important significance. In bronchiectasis over cavity there are large moist râles, mucous clicks and gurgles; they are larger than could occur if the lumen of bronchi in that region was normal. In chronic bronchitis the size of the râle corresponds to the normal size of the tube.

Gangrene and Pulmonary Abscesses.—In pulmonary gangrene and pulmonary abscess there may be one or more cavities. When single, they generally follow some inflammatory condition of the lung or adjacent viscera. The signs of cavity formation are limited to a small portion of the lung, and are more likely to be close to the surface. The patient is acutely ill. Respirations are rapid and motion markedly interfered with over affected area. Pulse is rapid, feeble and dicrotic. Before the stage of excavation there is dullness on percussion; vocal fremitus is increased; there is bronchial breathing. With the production of the cavity, the hollow, blowing, cavernous or amphoric quality of breath sounds will be associated with the signs of more or less complete consolidation. When the cavities are multiple, they are nearer to the surface of the lung than in bronchiectasis; there is early involvement of the pleura over affected area.

In bronchiectasis the patient is usually well nourished, and does not give the impression of being acutely sick. Changes in respiratory movement are most marked over lower third of lung.

MICROSCOPICAL EXAMINATION.—Presence of elastic fibres in the sputum is diagnostic of the destructive process of gangrene and abscess. They are absent in simple bronchiectasis.

Localized Empyema Opening into a Bronchus.—Localized empyema opening into a bronchus, with localized pyo-pneumo-thorax, may simulate a bronchiectatic cavity, and is differentiated by the following: The enlargement of the chest is localized at the site of the lesion. The percussion note is tympanitic when cavity is empty. If it contains fluid, there will be zone of flatness, which changes with the position of the patient. Lung fistula sound is present rather than the hollow, blowing or cavernous sound of bronchiectasis.

ACUTE CONGESTION OF THE LUNG.

Active hyperæmia of the lung may be general or local. It depends on *(a)* cardiac overaction due to muscular exertion, cardiac stimulants, nervous or emotional causes; *(b)* irritation of the respiratory tract due to inhalation of steam, extremely cold air or other irritants; *(c)* determination of blood to the lungs by chilling of the surface of the body or during the chill of acute diseases, especially malaria; *(d)* it occurs during onset of all inflammatory diseases of the lung.

Physical Signs.—The physical signs will vary according to the degree of congestion. The distension of the blood-vessels is especially marked in the alveolar walls, causing the pulmonary tissue to be increased in amount and less elastic, with corresponding diminution in the amount of tidal and residual air.

INSPECTION.—Dyspnœa, with respiration more rapid than normal.

PALPATION.—Vocal fremitus unaltered or slightly increased.

PERCUSSION.—Resonance slightly impaired; pitch somewhat raised; change in percussion note not sufficient to give well-marked dullness.

AUSCULTATION.—The respiratory murmur has less of the vesicular quality than normal; inspiration is harsher and slightly higher pitched; expiration is prolonged, higher pitched than inspiration, with a blowing quality. When localized, the above changes in the physical signs are more noticeable by comparison with the surrounding normal lung.

PASSIVE CONGESTION OF THE LUNG.

Passive hyperæmia of the lung is caused by interference with pulmonary circulation, due to *(a)* organic lesions at the valvular orifices or to pressure of interthoracic tumors; *(b)* to insufficiency of cardiac power, dependent upon myocardial disease or secondary to adynamic conditions, as typhoid fever, anæmia, etc. Feebleness of respiratory movements favors stasis at the borders of the lung, while gravity causes the circulation to be weakest in the most dependent portions (hypostatic congestion), which varies with the posture of the patient.

Long continuance of passive congestion causes exudation of serum into the interstitial tissue (hypostatic pneumonia), into the alveoli, infundibula and bronchi (pulmonary œdema), and red-blood cells and connective tissue elements into the stroma of lung (brown induration).

Physical Signs. INSPECTION.—Dyspnœa; respiratory movements most marked over upper portion of chest; loss of motion over base of lungs; slight cyanosis, especially during sleep and on exertion. Patient gradually assumes the sitting posture.

PALPATION.—Vocal fremitus normal over upper portion of lung; diminished along edge of lung and over base in simple, passive congestion. When secondary changes (induration) occur, vocal fremitus is increased.

PERCUSSION.—Upper portion of chest normal or hyper-resonant; over base, slight dullness, most marked over right side below angle of scapula. Secondary changes in the lung causes the dullness to be replaced by flatness.

AUSCULTATION.—Over upper portion of chest, the breath sounds may be normal; or with inspiration harsher and expiration blowing and prolonged. Over the lower portion of the chest, the vesicular murmur is feeble or absent. When induration occurs, the breath sounds have a bronchial quality. Râles are present in œdema.

In the early stage the physical signs are bilateral; later they become more intense, according to the posture of the patient, over certain portions of the lung.

PULMONARY ŒDEMA.

Pulmonary œdema is never a primary condition, but always secondary to *(a)* acute inflammation of the lung, *(b)* passive congestion, *(c)* or is part of a general œdema. The œdema will be local or general, according to the cause, and its distribution will be influenced by the same factors that were mentioned under passive congestion.

Physical Signs. INSPECTION.—The changes in size, shape and motion will depend upon the condition to which the œdema is secondary. Filling of the alveoli at the most dependent portion causes loss of motion, with compensation elsewhere.

PALPATION.—Vocal fremitus is diminished or absent over the lower portion of the chest. Rhonchi may be detected when fluid is present in the larger tubes.

PERCUSSION.—Dullness in the early stage over the base of the lung. As the air spaces become filled with fluid, dullness becomes more intense, and may be replaced by flatness. The line of dullness gradually extends upward with increased severity of the condition.

AUSCULTATION.—The distinctive physical sign of pulmonary œdema is the presence of fine, crepitating, moist râles. In mild cases these are heard over the most dependent portions and along the borders, while over the rest of the lungs the breath sounds may be normal. As the œdema becomes more intense and involves the bronchi, râles of larger size and more bubbling are also heard.

LOBULAR PNEUMONIA.

Synonyms.—Broncho-pneumonia, disseminated catarrhal pneumonia, pulmonary catarrh.

Lobular pneumonia is characterized by a filling of the alveoli by inflammatory products, which differ from those of lobar pneumonia in containing less of the fibrin factors of the blood and more of the elements drawn from the lining membrane of the alveoli and bronchi.

The pathological changes of lobular pneumonia may be due to a variety of causes: *(a)* Capillary bronchitis, *(b)* acute pulmonary catarrh of whooping cough and other infectious diseases; *(c)* passive or hypostatic congestion, *(d)* pulmonary infarction, *(e)* diffused tubercular infection. (See Pulmonary Tuberculosis.)

The distribution of the areas affected by catarrhal pneumonia will vary according to the nature of the cause; the type is determined by the physical signs. The affected lobules may be widely and fairly evenly scattered through both lungs, and separated by normal, emphysematous or collapsed lobules, giving (A) the disseminated type of lobular pneumonia. Usually a number of adjacent lobules are involved, and produce well-marked patches of consolidation in different portions of the lung. (B) The confluent type is due to the coalescence of these patches, causing one or more lobes to be almost completely consolidated, producing a condition which gives physical signs almost identical with those of lobar or croupous pneumonia.

Physical Signs. A. Disseminated Lobular Pneumonia.—The physical signs of this form have been described under D. Acute Bronchitis.

B. Confluent Lobular Pneumonia.—The physical signs differ from those of the disseminated type, as follows:

INSPECTION.—Dyspnœa is more markedly obstructive during inspiration. As both lungs are not equally involved, deficient expansion is more marked over one side of the thorax. When due to passive or hypostatic congestion, loss of motion is most marked over the base or dependent portions.

PALPATION.—Vocal fremitus may be increased when the areas of consolidation are close to the surface. When they are separated from the surface by emphysematous lung or surrounded by collapsed lobules, vocal fremitus may be normal or diminished.

PERCUSSION.—In the early stage percussion may be normal. When surrounded by collapsed lung, the percussion note is dull, but of a tympanitic quality. With increase in consolidation, the resonance is diminished, the note becomining dull and high pitched.

AUSCULTATION.—The change in the breath sound will vary with the amount of pulmonary consolidation and its relation to the surface of the chest. When the consolidated area is deep seated or surrounded by normal, emphysematous or collapsed lung, the bronchial quality will not be heard, or it will be faintly detected only at the end of expiration (see Fig. 24). The nearer the pneumonic patch approaches the surface, the more distinctly will the bronchial breathing be heard. The râles heard over the consolidated part differ from those heard in bronchitis, in being more uniform in size; their number is fairly constant, and the quality is clearer, higher pitched and more definitely ringing, resembling the crepitant and subcrepitant râles of lobar pneumonia.

Differential Diagnosis. Pulmonary Collapse (Atalectasis.)—Pulmonary collapse and lobular consolidation are both common in extensive bronchitis of the smaller tubes, and are usually associated. The physical signs will vary according as one or the other condition is predominant.

INSPECTION.—In both conditions motion is restricted, especially over the lower portion of the chest.

PALPATION.—In consolidation vocal fremitus is normal or slightly increased. In pulmonary collapse vocal fremitus is diminished or absent.

PERCUSSION.—In pulmonary consolidation the percussion note is dull. In pulmonary collapse there is diminished resonance, with tympanitic quality.

AUSCULTATION.—In consolidation the breath sounds are more or less bronchial in character. In pulmonary collapse the breath sounds are feeble or absent. Vocal resonance, in consolidation increased; in collapse, feeble or muffled.

Acute Disseminated Tuberculosis.—Differential diagnosis between acute disseminated tuberculosis with softening and disseminated lobar pneumonia is extremely difficult. Physical signs may be identical, the location of the two conditions being frequently the only distinctive point. Tuberculosis affects especially the apices, while lobular pneumonia involves the lower lobes, especially the base.

Acute miliary tuberculosis without softening is differentiated from broncho-pneumonia in that it involves the apex, and râles are not common.

Broncho-pneumonia involves the base, and is attended with râles.

Lobar Pneumonia.—Lobular pneumonia may be so extensive as to consolidate more or less completely an entire lobe. Lobular pneumonia, even when more or less lobar in character, does not present the uniform consolidation that occurs in lobar pneumonia, nor is it limited to one lobe or to one lung. In the second stage of lobar pneumonia over the affected portion of the lung the breath sounds are usually clear and unattended with râles.

LOBAR PNEUMONIA.

In lobar or croupous pneumonia the alveoli and smaller bronchi are filled with an exudate composed of coagulated fibrin and cellular elements. During the course of the disease the pathological changes that occur pass through three definite stages.

1. **Stage of Engorgement.**—The lungs are intensely congested, especially in the affected lobe, whose distended blood-vessels interfere with its expansion and elasticity; the pulmonary tension is altered, and there is slight enlargement of the lobe. There is more or less liquid secretion in the alveoli and bronchioles, and also œdema of the subpleural and pleural tissues. There may also be a layer of plastic material on the free surface of the pleura. This stage may last from a few hours to three or four days.

2. **Stage of Red Hepatization, or Consolidation.**—In this stage there is distension of the alveoli and bronchioles,

with serum, leucocytes and red-blood corpuscles. The serum coagulates and holds in its meshes of fibrilated fibrin the corpuscular elements. The consolidated portion is airless and "in a state of immovable expansion more densely solidified than it could be by any artificial injection with coagulable fluid." (Powell.)

The pleura over the consolidated portion is covered with a layer of the same coagulated exudate. In addition, the pleural sac may contain fluid serum, which may be clear, turbid, blood-stained or purulent. Complete consolidation of the affected lobe may occur in a few hours, or, beginning in a circumscribed portion, it may be days before it extends throughout the entire lobe; or it may remain limited to a portion of the lobe only. This stage lasts usually from five to seven days, terminating with the crisis.

3. State of Gray Hepatization, or Resolution.—The changes that take place during the stages of engorgement and consolidation are fairly constant and rapid. Those that occur during the third stage may be variable. At the beginning of the stage of gray hepatization the bulk of the lung is greater than in that of red hepatization, but it is less firm. The solid coagulum in the alveoli undergoes liquefaction; air again enters the alveoli, passing through the liquid exudate, giving rise to large moist râles that are named râle redux. The third stage of pneumonia may terminate in *(a) resolution.* The contents of the alveoli are removed by absorption and expectoration; the lung regains its resiliency and returns to normal condition. In the majority of cases perfect resolution and recovery occurs.

(b) Purulent Infiltration and Abscess of the Lung.—In this condition the coagulated fibrin liquifies, but, instead of fatty degeneration and absorption of the corpuscular elements, a suppurative inflammatory process continues; there is destruction of the lung tissue and the formation of cavities of varying sizes.

(c) Gangrene results from failure of nutritive circu-

lation, either through thrombosis of the large vessels or capillary stasis.

(d) Unresolved or Chronic Pneumonia.—In this condition the changes that ordinarily occur with the crisis are absent. There is a proliferation of new cells in the tissues of the lung and also in the alveoli. These organizes into new tissue, and there is a gradual development of fibrous tissue in the alveoli.

Physical Signs.—The physical signs of pneumonia have been divided into three stages, corresponding to the pathological changes in the lung. It must be constantly borne in mind that these pathological divisions are largely arbitrary, and that as the transition of one stage into the other may be rapid or gradual, so the physical signs of one stage merge into those of the succeeding stage.

INSPECTION.—*First Stage.* Patient lies on the affected side; circumscribed flush (pneumonic spot) over one or both malar bones; repiration rapid, 25 to 40, panting in character, especially during expiration. Motion on affected side slightly restricted. When the pleura is not involved, the movements of the two sides may be equal. When pain (pleurisy) is present, motion is less on the affected side than on the opposite; inspiration is either catching or restrained; expiration is slow. Movements of the opposite side are exaggerated.

Second Stage. Face dusky; marked movements of the nares; breathing rapid and panting. Slight enlargement of the affected side of the chest. Motion on the affected side diminished, with compensatory movement on the opposite side. Breathing will be costal or abdominal, according as the upper or lower lobes are involved. Involvement of the diaphragm (diaphragmatic pleurisy) causes absence of abdominal breathing.

Third Stage. With the absorption of the exudate, movements of the affected side gradually return to normal.

PALPATION.—*First Stage.* During the early stage of engorgement vocal fremitus is normal or slightly diminished. As consolidation occurs, it gradually increases in intensity.

Second Stage. When consolidation of the lung is complete, vocal fremitus is greatly increased. In central pneumonia vocal fremitus may be but slightly increased. It is diminished in marked thickening of the pleura, and absent when effusion occurs in the pleural sac, and also when the large bronchi leading to consolidated portion is blocked. Friction fremitus may be felt over the affected part in the earlier stages of consolidation, while movement of the lung is still possible.

Third Stage. There is gradual diminution of vocal fremitus with return to the normal. During the early stage of resolution, blocking of the bronchus is most likely to occur, with a temporary absence of vocal fremitus. After paroxysms of coughing vocal fremitus may return. Absence of vocal fremitus after the removal of exudate from the alveoli is due to exudation on the pleura or filling of the pleural sac.

PERCUSSION.—*First Stage.* Percussion note at first may show no change. As congestion increases, the intensity diminishes; it is shorter in duration, higher pitched and resonant, but with a slight tympanitic quality. The dullness of consolidation is most apt to appear first just beneath the angle of the scapula and to develop towards the axillary line. As air in the alveoli is replaced by the solid exudate, the note becomes duller, higher pitched and less resonant, until complete consolidation.

Second Stage. The percussion note is dull and high pitched, but even in complete consolidation resonance is present in a very slight degree. The sound is flat if pneumonia is complicated by extensive pleuritic changes.

When consolidation of the lobe is not complete, the air-containing, but relaxed, lobules scattered through the consolidated portion cause the percussion note to have a dull, tympanitic or tubular quality. When the pneumonia is central and air-containing lung is interposed between the chest wall and the consolidated portion, the character of the percussion note will depend upon the condition of the interposed lung. *(a)* When the lung is in a state of compensatory emphysema and tension is increased, the percussion note gives increased resonance. *(b)* When the lung is relaxed, the percussion note is dull, with tympanitic or cavernous quality. Percussion dullness is especially apt to be absent in children and in old persons, due to the elasticity of the chest wall in children and the emphysematous condition of the lung, associated with rigid thorax, in the aged.

The normal boundaries of the lobes are changed, so that when the lower lobe is involved the dullness extends higher than the normal outline, and may be detected almost to the upper portion of the scapula. When the upper lobe is involved, the dullness is most marked anteriorly. Enlargement of the consolidated portion allows of relaxation of the unaffected portions of the lung on the same side, over which a peculiar tympanitic resonance is obtained—Skoda's resonance. Percussion over the opposite lung shows exaggerated percussion note, due to compensatory emphysema.

Third Stage. As the removal of the exudate continues and the alveoli again contain air, the percussion sound becomes more resonant. The pulmonary tissue does not at once regain its normal elasticity, and frequently with the return of resonance the sound has a more or less tympanitic quality and is low pitched, on account of the relaxation of the lung. Dullness may be continued after resolution is well advanced, owing to the presence of pleural exudate. Flatness over the base of the lung occurs in cases of unabsorbed effusion in the pleura or the occurrence of empyæma. It was noted above that lobar pneumonia in children is frequently unattended with marked dullness. The presence of well-marked dullness, with increased resistance over the lower portion of the lung, is usually due to the occurrence of acute empyemia during the course of a pneumonia.

AUSCULTATION.—*First Stage.* In the earliest stage of engorgement the breath sounds are weaker than normal over the affected portion. Over adjacent portions the breath sounds are slightly exaggerated; later, as the engorgement increases, the breath sounds become harsher, especially marked during expiration. As the exudation fills the alveoli, the inspiratory portion of the breath sound loses its vesicular element, has a faint blowing quality, and the pitch becomes higher. The expiratory portion is at first slightly prolonged, higher pitched, with a distinct bronchial character. As the consolidation becomes more marked, there is a gradual disappearance of the normal vesicular murmur and an increase in the intensity of the bronchial breathing.

The crepitant râles (râle indux), which has been considered by some authors as characteristic of this stage of pneumonia, may be present before percussion dullness or the occurrence of the bronchial type of breathing. The crepitant râle is not always present. It may be absent *(a)* when the consolidation is central and there is no pleural involvement; *(b)* when complete consolidation occurs very rapidly; *(c)* in secondary pneumonia. When pneumonia is superadded to acute bronchitis, large and small moist râles may be present and mask the crepitant râle and the early changes in the breath sounds. The crepitant râle, when present, is of important diagnostic significance.

Second Stage. Bronchial breathing during the second stage is present in proportion to the degree of consolidation. As the alveoli becomes filled, the breathing passes from a stage of high-pitched, prolonged expiration, broncho-vesicular, to pure tubular and laryngeal breathing. The bronchial, tubular and tracheal breathing that is present in pneumonia differs from that heard over the bronchi, trachea and larynx in the normal lung. It is higher pitched; expiration is more prolonged, and the tubular element has a peculiar, distinct, "audible" quality, which is a striking feature of the sound. Although it may not be very loud, this quality of the sound is easily detected when present. This distinctive quality of the breath sound in pneumonia is due to the resonating influence of the dense consolidated lung on the vibrations brought to it by the walls of the bronchi. In central pneumonia the interposed unconsolidated lung causes the breath sounds to have a combined distinct bronchial breathing, associated with normal vesicular murmur. The bronchial element in these cases is especially marked during expiration. When the amount of consolidation is slight and situated deep in the lung, the bronchial sound may not be detected as such, but a high-pitched, blowing sound may be heard after the expiratory sound of the normal lung has ceased. In cases of central pneumonia, increased vocal resonance (bronchophony) may be the only sign present. Plugging of a large bronchus causes the bronchial breathing to be absent. This is especially apt to occur in the massive type of pneumonia. Thick exudation upon the pleura or effusions into the pleural cavity gives a weakening or absence of the bronchial sounds. Effusions into the pleural cavity, especially where they occur before the stage of complete consolidation, give a type of breathing that is similar to cavernous breathing. In pneumonia affecting the upper lobes of the lung, feebleness or absence of bronchial breathing may occur without blocking of the bronchus or the interference of thick pleural exudate. Over the unaffected portion of the same side, the respiratory sounds may be feeble or exaggerated, according as the lung is relaxed or is in a state of compensatory emphysema. Over the opposite lung the breath sounds are exaggerated. Frequently bronchial breath sounds may be transmitted to the opposite side, suggesting the occurrence of double pneumonia. Vocal resonance is increased in intensity in proportion to the degree of consolidation, and corresponds to the various changes that occur in the bronchial breathing. Pectoriloquy is present in complete consolidation. In central pneumonia, when uninvolved overlying lung masks the physical signs, increase in vocal resonance may be the only diagnostic symptom present; it should always be sought for in doubtful cases, especially in the aged.

Third Stage. The earliest indication of change in the consolidated lung is that the peculiar, clear, "audible" character of the bronchial breathing of the second stage is lost. The breath sounds become bronchial, and their vibratory quality gradually disappears. With these changes in the bronchial sounds, air again enters the bronchioles and alveoli, and medium-sized râles (râle redux) are heard with inspiration and expiration. As the alveoli become free of the exudate, the moist râles diminish in number and finally disappear. The vesicular quality returns as the bronchial is lost.

As the lung does not at once regain its normal elasticity, the expiratory sound may for a variable time be slightly prolonged and low pitched. In chronic unresolved pneumonia the signs of the secondary stage persist for a long time, and are gradually replaced by those of chronic interstitial pneumonia. When the pneumonic area undergoes purulent infiltration, the râle redux is associated with or displaced by larger liquid râles; the bronchial element of sound persists, and later, with the destruction of lung tissue, cavernous breath sounds are present.

Vocal Resonance.—With the beginning of the third stage, pectoriloquy of the second stage is replaced by bronchophony, which gradually diminishes in intensity until it becomes exaggerated resonance. With the completion of resolution, the voice sounds return to the normal.

Differential Diagnosis. Acute Pulmonary Congestion.—During the early part of the first stage of pneumonia the harsh respiratory murmur over the lung is due to acute congestion of the entire respiratory tract. As the localization of the congestion occurs in the lobe that is to be affected, there will be a gradual increase in the harshness of the respiratory murmur in comparison with the rest of the lung.

Pleurisy with Effusion.—During the stages of consolidation and resolution, pneumonia may be confounded with effusion into the pleural cavity. It is especially important that these two conditions be differentiated, on account of the frequency with which empyema follows pneumonia, especially in children.

INSPECTION.—In pneumonia the size of the chest is only slightly increased; the intercostal spaces persist, not being flattened or bulged; motion of the affected side is limited; of the opposite, slightly increased. In effusion into the pleura the enlargement of the affected side is marked; there is flattening or bulging of the intercostal spaces. The motion over lower portion of the chest is absent. *Position of the heart:* In pneumonia the apex beat is in a normal position. In effusions the heart is displaced. Location of the apex beat of the heart is one of the most important signs in separating the two conditions.

PALPATION.—In pneumonia the vocal fremitus is markedly increased, except when the bronchus is plugged. In effusion vocal fremitus is absent, and is only detected when adhesions are present. In pneumonia the diaphragm is not displaced downwards, and there is no change in the position of the abdominal organs. In effusion there is marked displacement of the diaphragm; the liver or spleen are palpable below the free border of the ribs.

PERCUSSION.—In pneumonia the note is dull, but still resonant; it is not flat. In effusion, note flat or toneless. In pneumonia the area of percussion dullness follows the anatomical division of the lung. In effusion it follows a curved line, being highest in the axilla. Skoda's resonance may be present in both conditions.

AUSCULTATION.—In pneumonia the breath sounds over area of dullness are bronchial or tubular in character. The sound gives the impression of being produced close to the surface. In effusion the breath sounds are absent over the lower portion of flatness; may be present at the upper level of the fluid, and differ from those of pneumonia in lacking clearness and being more or less distant from the surface. In pneumonia during the stage of resolution, râles are present. In effusion, liquid râles are not detected; friction râles may occasionally be heard.

Vocal Resonance.—In pneumonia, bronchophony or pectoriloquy is present. In effusion over lower portion of thorax, the voice sounds distant from the ear. Over the upper portion of the fluid pectoriloquy of a peculiar nasal character is heard (ægophony).

Hemorrhagic Infarction.—In hemorrhagic infarction the consolidation of the lung is due to the presence in the alveoli of the affected area of coagulated blood, so that the physical condition of that portion of the lung involved by hemorrhagic infarction is identical with that of a localized lobar pneumonia. Differential diagnosis is based on the position and distribution of the consolidation and the presence of valvular disease of the heart. In pneumonia the entire lobe is usually involved. Hemorrhagic infarction is localized, and is generally most apt to occur at the lower portion of a lobe and along its border. In pneumonia, unless complicated by organic cardiac disease, there are no adventitious heart sounds. Hemorrhagic infarction is generally associated with the physical signs of valvular lesion of the heart. The dyspnœa in pneumonia is panting; the patient lies upon the affected side or more or less flat upon the back. In hemorrhagic infarction the patient assumes the position of orthopnœa during the early portion of the attack.

Collapse of the Lung.—When this condition occurs in children, or when it affects the entire lobe, differential diagnosis may be very difficult. On inspection, in pneumonia, there is slight enlargement of the side. In collapse, retraction of the side occurs. In pneumonia there is increased vocal fremitus. In collapse, fremitus is diminished or absent.

In pneumonia there is dullness on percussion. In collapse the resonance is slightly impaired and associated with marked tympanitic quality.

In pneumonia there is well-defined bronchial breathing. In collapse the breath sounds are feeble; may have slight bronchial character, but are indistinct and distant from the ear. Pneumonia, during first and third stages adventitious sounds present. Collapse, these signs may be absent.

CHRONIC INTERSTITIAL PNEUMONIA.

Synonyms.—Cirrhosis of the lung. Fibroid phthisis.

The distinctive pathological change in this type of pulmonary disease is the increase in the fibrous structures of the lung. This overgrowth of connective tissue may be limited to the peribronchial tissue of the larger or medium-sized tubes. It may affect the interlobular septa, extending into the interlobular wall. In other cases it primarily involves the walls of the alveoli themselves, while in others it starts in the subpleural and pleural tissues and gradually extends inwards. It is rare to find fibroid induration limited to the different tracts above mentioned, although it may have its origin in one or the other of them.

The effect of increase of fibrous tissue is *(a)* to increase the solid structures of the lung. *(b)* According to the law of fibrous tissue, contraction takes place. *(c)* The induration of the tissue and the thickening of the walls of the alveoli interfere with the elasticity of the lung, and causes more or less impairment of the pulmonary function. *(d)* Following the contraction, there is more or less atrophy of the alveolar walls, resulting in emphysematous dilatation, or disappearance of the walls, and coalescence of adjacent alveoli. Bronchiectasis of varying degree is also present.

Three types of pulmonary fibrosis can be recognized by physical signs: (1) The massive or lobar type; (2) the broncho-pneumonic type; (3) the disseminated reticular or diffuse form. The type of the disease corresponds more or less closely to the etiological factors.

(1) Lobar pneumonia and the confluent type of broncho-pneumonia with collapse are often the origin of the massive or lobar type. Pleurisy with effusion, in which there has been extensive changes in the pulmonary subpleural tissue and long retention of fluid in the sac, causing more or less pulmonary collapse, is frequently followed by a lobar type, in which the primary changes are at the surface of the lung, crippling its respiratory function, and followed by retention of secretion within the alveoli, secondary broncho-pneumonia, and subsequent diffuse fibrosis. The occlusion of a large bronchus by foreign bodies or by pressure, as of aneurism, causes the tissue beyond the obstruction to collapse more or less. There is retention of inflammatory exudates, and the subsequent development of fibroid tissue.

(2) The broncho-pneumonic type may be induced by those conditions which cause diffuse capillary bronchitis or broncho-pneumonia. The broncho-pneumonic type, when localized at the apices, is especially prone to occur as a part of the pathology of pulmonary tuberculosis, and is to a large extent a conservative process. It may occur either at the beginning of the disease, may follow on acute secondary inflammation or may be cicatricial when there has been cavity formation.

(3) The disseminated type is generally dependent upon chronic bronchial irritation which may follow simple bronchitis, or may be due to the mechanical irritation in the so-called dust disease, or pneumono-

koniosis. Two forms of diffuse fibrosis are recognized by physical signs: 1. The emphysematous form, in which the fibroid tissue of the peribronchial and interlobular portions of the lung are especially involved and there is associated dilatation of the alveoli. 2. The contracted form, in which the alveolar walls and the subpleural tissue are especially involved. In this there is marked loss of the expansile tissue of the lung.

Physical Signs. INSPECTION.—In the localized fibrosis of the lobar and broncho-pneumonic types there is marked retraction of the chest over the seat of the disease, the extent being in proportion to the amount of lung tissue involved. Retraction of the lung causes a flattening of the chest wall, raising of the diaphragm and displacement of the mediastinum and heart toward the affected side. Change in the position of the ribs causes depression of the shoulder and curvature of the spine. The respiratory movements over the affected area are restricted in proportion to the extent of the disease. The opposite side will be in a state of compensatory emphysema and functional activity.

Diffuse Type.—In the emphysematous form the shape and size of the chest and the respiratory movements will correspond to those described under Large Lung Emphysema (see Emphysema). In the contracted form the shape of the chest is more or less analogous to the small lung or senile form of emphysema. There is contraction of the lower portion of the thorax; the intercostal spaces are narrowed; the ribs may overlap. Expansion of the lung is deficient; the diaphragm is carried upward, and the supraclavicular and suprasternal spaces are deepened. It is rare for this condition to be uniform or equally marked on both sides. In tuberculosis one entire lung may be involved, while in the other it may be limited to the apex. When secondary to mechanical bronchitis, the changes are more uniform.

PALPATION.—Vocal fremitus is variable, and gives important information as to the nature of the change in the lung and the extent of the involvement. In the massive or lobar type there will be increase in vocal fremitus. In the broncho-pneumonic type the change in vocal fremitus will correspond to the extent of the disease and its nearness to the surface of the lung. The vocal fremitus may be feeble or absent when there is obstruction of the bronchi leading to the affected area, and when there is marked thickening of the pleura. In the diffuse type vocal fremitus is markedly diminished in the emphysematous form. In the contracted form it may be increased, but usually, on account of its association with pleural thickening, it is feeble or absent. The localization of the disease also causes variations in the intensity of the vocal fremitus. Fibrosis of the apex is usually attended with increased vocal fremitus, while when the base is involved the vocal fremitus is diminished or absent.

PERCUSSION.—Increase of the solid structure of the lung causes dullness of the percussion note. Loss of tension of the lung and diminution in the size of the alveoli causes the dullness to have a peculiar wooden and tympanitic quality. Thickening of the pleura causes a more marked dullness, with increased sense of resistance. In the emphysematous form the increase in the size of the alveoli causes the percussion note to be hyper-resonant.

AUSCULTATION.—In the lobar type the breath sounds are bronchial in character and more or less weakened, but differ from those present in true lobar pneumonia in being less intense and having a somewhat soft, blowing quality. Bronchophony is also present, but is weak and distant.

In the broncho-pneumonic type, if the area involved is close to the surface of the lung, bronchial or broncho-vesicular breathing will be present. When situated deeper in the lung and covered by distended alveoli, bronchial breathing may be absent, and the respiratory murmur will be feeble. When râles are present over the affected areas, they will have a sharp, metallic quality. In the disseminated type one of the characteristic features is the feebleness or absence of the vesicular element of the inspiratory sound, associated with feeble, prolonged and slightly bronchial expiratory sound. Pleural changes cause both the vesicular and bronchial elements of the breath sounds to be weakened or obliterated.

Differential Diagnosis.—The differentiation of the fibrosis that occurs in tuberculosis from that of nontubercular diseases of the chest is frequently impossible. Fibrosis always occurs in certain forms of tubercular infection of the lung, and has no distinctive features. The condition known as coal miners' phthisis, fibroid phthisis, etc., is frequently due to the combination of dust irritation and tubercular infection. Frequently the fibroid changes in the lung are so marked as to become the most important factor, while the tubercular changes are subsidiary.

EMPHYSEMA.

Pulmonary vesicular emphysema is an overdistension of the air-vesicles, and may be acute or chronic. The varieties of emphysema are: (1) Compensatory or vicarious. This may be limited to a few lobules, to a lobe or involve one lung only. This type of emphysema occurs when distension of a portion of the pulmonary tract is compensatory for deficient expansion in other portions of the lung.

(2) General vesicular emphysema. In this variety, associated with dilatation of the air-vesicles, are structural changes in the walls of the alveoli which diminish their resiliency and lead to atrophy. Two types of general emphysema are recognized: *(a)* Chronic, large lung or hypertrophic emphysema, and *(b)* small lung, atrophic or senile emphysema.

(3) Interlobular or interstitial emphysema, with infiltration of air into the pulmonary stroma.

In general, chronic, large-lung emphysema the most marked change, in addition to increased dilatation of the alveoli, is a loss of elasticity of the alveolar walls, which influences the respiratory function. As has been mentioned before, while the pulmonary tissue is passive during inspiration, merely dilating with enlargement of the thorax, during expiration it is active, and plays a most important part in contracting the chest. Perfect respiration requires that the chest be contracted as well as expanded. The power of the lung to contract is due to *(a)* elasticity of the alveoli; *(b)* contraction of the muscular tissue of the bronchial tract.

In proportion as the normal elasticity of the pulmonary tissue is impaired in emphysema, the following conditions are induced: *(a)* Faulty expiratory contraction leads to gradual overdistension of the lungs, increase in the amount of residual air and diminution in the amount of tidal air. The chest wall, unacted upon by the elasticity of the lung, assumes at first the position of full inspiratory expansion. As the ventilation of the lung is imperfect, dyspnœa is easily induced on exertion, and the accessory inspiratory muscles are called into play to increase still further the capacity of the chest. The upper portion of the bony thorax is carried upward; the sternum is carried forward; ribs rotated outwards; the intercostal spaces widen; antero-posterior diameter of the chest increases. "Enlargement of the chest to the limit of thoracic resiliency occurs." (Powell.) Enlargement of the lung displaces the heart towards the median line, and the diaphragm is depressed, together with the abdominal organs.

(b) Circulatory changes. The circulatory system is affected in two ways: (1) Normally the elasticity of the lung causes a negative pressure within the thorax,

which, during inspiration, equals 7 to 9 mm. of mercury. "The elastic fibres of the lung are upon the stretch and are pulling upon the ribs, intercostal spaces, upon the diaphragm and upon the heart and great vessels. The elastic pull of the lung . . . assists the diastolic expansion of the ventricles . . . and acts upon the vena cava within the chest and generates within them, as well as within the right auricle, a force of suction. This suction from within the chest extends to the great veins just without it in the neck."[1]

As the elasticity of the pulmonary tissue becomes impaired, the suction force of the lung decreases; there is a corresponding imperfect dilatation of the right auricle and emptying of the great veins. This interferes with the return circulation, which early shows itself in distension of the jugular veins and a tendency to general venous stasis.[2] (2) The dilatation of the alveolar walls, with the subsequent atrophy, causes obstruction to the pulmonary circulation through narrowing the capillaries, by stretching and by obliteration. The effect of this circulatory obstruction is to raise the pressure in the pulmonary artery, which is early indicated by accentuation of the pulmonic second sound. To overcome the increased tension in the pulmonary artery, the right heart hypertrophies; sooner or later the hypertrophy fails to keep pace with the progressive obstruction of the pulmonary circulation and the increased work. The right ventricle is unable to empty itself; dilatation of the cavity occurs, with incompetency of the tricuspid valve, and regurgitation in the right auricle. When tricuspid regurgitation occurs, the imperfect venous return, due to diminished negative pressure, is acutely increased. Pulsations occur in the jugular vein; there is passive congestion of all the abdominal viscera, with enlargement of the liver and spleen, the occurrence of ascites and general anasarca.

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[1] American Text-Book of Physiology, Vol. I, p. 95.

[2] In compensatory emphysema the decrease in negative pressure is not present. In the senile type it is not a marked feature of the disease.

Physical Signs.—The physical signs of emphysema vary with the degree of pulmonary distension and loss of elasticity. They are modified by intercurrent attacks of acute bronchitis, spasm of the bronchi (asthma) and the secondary changes in the circulatory system.

INSPECTION. *Large-Lung Emphysema.*—The patient presents a more or less characteristic appearance. The chest is enlarged in all directions, especially in the antero-posterior diameter. The dorsal curve of the spine is exaggerated. The sternum is carried forward; the ribs are rotated outward and are more horizontal; the upper intercostal spaces are widened, and the lower portion of the chest is seemingly contracted in comparison with the upper portion, but there is an increase in the transverse diameter. These changes in the bony thorax give the barrel-shaped chest of emphysema. Mensuration shows enlargement of the thorax is in all directions, especially marked in antero-posterior diameter.

The movements of the thorax are altered; inspiration is short, jerky, with very slight expansion from first to fourth ribs. The muscles of the neck are hypertrophied and prominent, the upper portion of the chest being pulled upwards by the accessory muscles of inspiration. With inspiration the suprasternal, supraclavicular and upper intercostal spaces are deepened. The lower portion of the chest is depressed during inspiration by the action of the diaphragm, and breathing is chiefly abdominal.

Expiration is tardy, slow and prolonged, with forcible contraction of the abdominal muscles. During expiration the intercostal spaces may be even with, or project beyond, the level of the ribs. Cardiac impulse is not seen in the normal site, but is displaced downward and inward. Epigastric pulsation is marked. The jugular veins are prominent and, in tricuspid regurgitation, pulsating.

Small-Lung Emphysema.—The patient has the wasted, shrunken appearance of old age. The thorax is contracted, the shoulders are rounded, but the clavicles, ribs and sternum are depressed; the intercostal spaces are narrowed below the fourth rib. The lower ribs are very oblique, and the edges may be in contact. The movements of respiration are shallow; the thorax is more rigid than in the large-lung type. During inspiration there is deepening of the supraclavicular fossa and intercostal spaces. The diaphragm is not depressed, but its descent is limited. Cardiac impulse is not displaced downward and toward the median line, as in the large-lung type, but may be slightly elevated and carried further to the left. Epigastric pulsation and distension of the jugular veins are absent.

Compensatory Emphysema.—When compensatory emphysema involves the entire lung, the enlargement of the side is attended with movements of increased function, *i. e.,* increased expansion during inspiration, while in expiration the contraction of the thorax is energetic and prolonged. The cardiac position varies with the cause of the emphysema and the side involved.

Emphysema limited to a lobe or part of a lobe will affect the size, shape and movement of the thorax, according to the cause. When emphysematous dilatation occurs in portions of the lung surrounding areas of consolidation or induration, it may prevent the deformity that is usually associated with the primary condition.

PALPATION. *Large-Lung Emphysema.*—Vocal fremitus varies in proportion to the rarefaction of the lung and the diminution of pulmonary tension. It may be normal in mild cases, diminished or absent in well-marked. Cardiac impulse felt in the epigastric region is diffuse; the force depends entirely upon the degree of hypertrophy of the right ventricle. The liver may be felt below the free border of the ribs; the edges are firm, smooth and rounded in proportion to the congestion from disturbed venous return in the vena cava. The spleen may be enlarged and palpable, secondary to hepatic congestion. When emphysema is associated with bronchitis or spasm of the bronchi, rhonchi may be felt.

Small-Lung Emphysema.—Vocal fremitus is but slightly changed, and may show slight increase in intensity, especially over the areas of large bronchi.

Compensatory Emphysema.—Increased tension of the lung causes the normal vocal fremitus to be slightly increased.

PERCUSSION.—*Large-Lung Emphysema.*—Rarefaction of pulmonary tissue and increased amount of air contained in the thorax causes the percussion note to be hyper-resonant; the low tension of the pulmonary tissue lowers the pitch. The condition of the chest wall (page 84) permits the bony quality to be added to that of the pulmonary tissue, so that the quality is changed. According as these different elements enter into the percussion sound, it has been described as vesico-tympanitic, band-box, boardy, woodeny, etc. The character of the sound does not show variation during inspiration and expiration. The borders of the lung are extended, and resonance may be obtained as low as the twelfth rib behind. The normal areas of hepatic and cardiac dullness are diminished or absent.

Small-Lung Emphysema.—The percussion note is hyper-resonant, but the pitch is not lowered; the quality is clearer and more tympanitic than in the large lung type. The areas of cardiac dullness are not diminished, but may be increased by retraction of the pulmonary borders. Hepatic dullness is slightly higher than normal. The rigid chest wall gives an increased sense of resistance.

Compensatory Emphysema.—In acute cases all the elements of normal percussion sounds are exaggerated. The note is hyper-resonant, clear and slightly higher in pitch.

In long-standing cases, secondary changes in the lung may cause loss of tension and the percussion sound of the large-lung emphysema. In chronic localized emphysema the percussion note varies according to the conditions with which it is associated.

Auscultation.—*Large-Lung Emphysema.*—During inspiration the vesicular murmur is short, feeble, or may be inaudible, being replaced "by a low-pitched, rumbling sound of muscular contraction." (Fowler.) During expiration the breath sound is prolonged, and continues with nearly equal intensity to the end of the act. In direct proportion to the severity of the disease, the ratio of the length of inspiration to expiration is altered. Expiration becomes longer than inspiration, sometimes reversing the normal ratio, being four times as long as inspiration. The pitch is low, and the quality slightly blowing. When emphysema is associated with bronchitis and spasm of the bronchi, various-sized dry and moist râles are heard (spasmodic emphysema).

When emphysematous blebs have formed beneath the pleura, fine crackles are heard with inspiration and expiration, or both (fine crackling crepitations, emphysematous gurglings). With the failing right heart, fine, moist, crepitating râles, due to pulmonary œdema, are heard over the base of the lungs.

Cardiac sounds are not heard over the normal area, and vary according as hypertrophy or dilatation is most marked. Over the normal apex of the heart the first sound is low pitched and prolonged in hypertrophy, short and sharp when dilatation and cardiac weakness occur. Over the base the cardiac sounds are heard at a lower level than normal, and there is accentuation of the second sounds to the left of the sternum, due to increased tension of the pulmonary artery. A systolic murmur is frequently present over the displaced apex. At first this may be present only after exertion, or during transient attacks of intercurrent bronchitis. Later it may be permanent, with all the associated signs of tricuspid regurgitation. Cardio-respiratory murmurs are at times present over the normal area of cardiac dullness.

Small-Lung Emphysema.—The deviations from normal are not as marked as in the large lung. The inspiratory sound is nearly normal in length, but feeble. The expiratory sound is prolonged, but rarely exceeds the length of inspiration; is harsher, and its pitch is not as low.

Compensatory Emphysema.—Breath sounds are exaggerated. They retain their normal characteristics, but are pure in type.

Differential Diagnosis.—The distinctive signs of large-lung emphysema are bilateral enlargement of the chest, with increase in the antero-posterior diameter and rounded contour (barrel-shaped chest); widening of the intercostal spaces; restricted inspiratory movement, with prolonged and labored expiration; displacement of the apex beat and epigastric pulsation.

Feeble vocal fremitus, associated with hyper-resonant and low-pitched percussion note.

The inspiratory portion of the breath sounds is short, feeble or absent, while the expiratory portion is prolonged, low pitched and relatively more intense.

Pneumo-thorax.—Enlargement of the thorax is not symmetrical, but more marked over the affected side. The intercostal spaces of the affected side are obliterated. Movements of the two sides are unequal. There is absence of motion on the affected side, with active movement of the opposite. The heart is displaced toward the unaffected side. The percussion note is hyper-resonant, but of tympanitic quality, the normal vesicular quality being absent. Coin test shows a peculiar metallic quality of sound. Vocal fremitus is absent. Vesicular murmur is absent. Amphoric breathing is present on inspiration and expiration when the opening to bronchus is patent. Lung fistula sound at times detected.

Hydro-pneumo-thorax.—Below level of fluid there is flatness on percussion. On shaking the patient succussion sounds may be detected, with metallic tinkles.

Cardiac Dyspnœa.—In acute febrile diseases and in anæmia, physical signs similar to those obtained in a mild degree of emphysema are frequently present, especially the hyper-resonant, low-pitched percussion note. This is differentiated from true emphysema in that the respiratory sounds retain their normal relation to each other.

ASTHMA.

Asthma is a disease characterized by attacks of intense dyspnœa of a paroxysmal type, occurring more or less suddenly.

Attacks of asthmaic dyspnœa may be due to acute narrowing of the lumen of the bronchial tubes by *(a)* spasm of the muscles of the bronchi (idiopathic or true asthma), *(b)* vaso-motor dilatation of the blood-vessels or inflammatory swelling of the mucous membrane (secondary asthma).

Between the attacks in the primary type the respiratory tract may be normal. In the second type it shows the change of the primary condition.

Condition of Lungs During Asthmatic Attack.—In proportion to the intensity of the attack, there is increase in the amount of residual air and a corresponding decrease in the quantity of tidal air, with resultant overdistension of the alveoli. The chief factor in causing interference with the normal movement of the air during inspiration and expiration is the spasmodic contraction of the muscular tissue present in the bronchial tubes. Muscle fibres have been demonstrated in the bronchial tract as far as the infundibula. The muscle fibres of the bronchi are passive during inspiration, but during expiration their contraction is an important factor in forcing the air out of the lung and in the production of normal expiratory breath sounds.

During an asthmatic attack the normal rhythmical action of the bronchial muscles is disturbed. With expiration the contractions are excessive, narrowing the lumen of all the tubes, and even closing entirely the smaller ones, especially the non-cartilaginous. With the following inspiration active contraction of the muscle ceases, but more or less spasm remains, so that there is persistent interference with the movement of air in the lung; but it is not so great as during expiration. In mild attacks the inspiratory obstruction may not be present. With each respiratory act more air is taken in than is expelled, until the thorax is distended to the utmost; there is imperfect ventilation of the lung and deficient oxygenation of the blood, with attending symptoms of dyspnœa. The effect of an asthmatic attack is overdistension of the lung, causing depression of the diaphragm, enlargement of the thoracic cavity in all directions; the heart is displaced downward and toward the median line.

Contraction of the bronchi and overdistension of the alveoli cause diminution of the normal negative interthoracic pressure during inspiration, and may give rise to positive pressure during both inspiration and expiration, which in turn causes interference with pulmonary circulation, overdistension of the right ventricle, and interference with venous return circulation.

With the subsidence of an attack, the pulmonary condition may return to normal. Repeated and long-continued attacks of asthma gradually produces permanent dilatation of the air-vesicles and secondary emphysema.

Physical Signs. INSPECTION.—*(a) During attack:* The face may be pale or slightly cyanotic; the patient generally sits up in the position to give greatest leverage to auxiliary muscles of respiration; the shoulders are elevated; the chest assumes the barrel shape of emphysema. With inspiration, the muscles of the neck, especially the sterno-cleido-mastoid, are prominent, and cause the movements of the chest to be short and jerky, lifting the thorax *en masse,* but with very little expansion. Interference with the free entrance of air into the alveoli causes depression of the soft parts, so that the suprasternal, supraclavicular and intercostal spaces are deepened. The diaphragm is forced to the lowest point possible, the lower portion of the thorax and sternum are depressed, and the epigastric region becomes more prominent. Expiration follows inspiration without a pause, and is prolonged, labored, but feeble. Movement of thorax is very slight, and due to the action of the auxiliary muscles of expiration, which, forcing the bony thorax against the inflated lung, cause bulging of the intercostal spaces and supraclavicular fossæ. Respirations are not increased in frequency, and may be less than normal. Jugulars are distended, and the apex beat is displaced downward and to the right, with marked epigastric pulsation.

(b) Between the attacks: the condition of the lungs may be normal. When asthma has produced permanent dilatation of the air-vesicle, the shape and movement of the thorax are those of emphysema.

PALPATION.—*(a) During attack:* vocal fremitus never increased; may be diminished or absent. Over a given area it varies from time to time, according to the degree of contraction of bronchi leading to the part. Rhonchi may be felt. The cardiac impulse is displaced downward to the right, and is diffused; pulse is small and intermittent, especially during inspiration (pulsus paradoxus). *(b) Between the attacks:* vocal fremitus is normal or diminished, as in emphysema.

PERCUSSION.—*(a) During attack:* percussion sound hyper-resonant. During early stage of the disease, with increased tension in the lung, the sound is slightly higher pitched, but clear. After repeated attacks, the diminished elasticity of the lung causes the pitch to be altered (lower) and the quality to be tympanitic or boardy (see Emphysema). Enlargement of the lung causes cardiac and hepatic areas of dullness to disappear. The borders of lungs are extended, and show no variation between inspiration and expiration. *(b) Between the attacks:* resonance may be normal or more or less emphysematous.

AUSCULTATION.—*(a) During attacks:* inspiratory vesicular murmur may be heard from time to time. When present, it is of normal quality, but weak, short and jerky. Usually it is absent or obscured by sibilant or sonorous râles.

During expiration respiratory murmur is usually absent, being replaced by prolonged dry râles of sonorous, sibilant, cooing or whistling type. The râles are constantly changing in intensity and character over a given area, at times disappearing, either with return of normal vesicular murmur or absence of all sound. With subsidence of the attack, large and small-sized moist râles are present, due to secretion in the bronchi.

When asthmatic attacks occur during the course of acute or chronic bronchitis, the dry and moist râles are both present. Vocal resonance is normal or somewhat diminished; cardiac sounds during attack are masked by dry râles in the lung.

(b) Between the attacks: respiratory sound may return to the normal, or a few wheezing râles may be present for some time after dyspnœa has disappeared. After severe attacks, fine, bubbling, moist râles, with feeble respiratory murmur, may be heard for some hours or days over the base of the lung and along the borders. In chronic cases, emphysematous breathing is present between the attacks.

Differential Diagnosis. Cardiac Dyspnœa.—Subjects of valvular and myocardial disease of the heart may suffer from intercurrent attacks of spasmodic dyspnœa. It has the following physical signs which differentiate it from asthma:

INSPECTION.—The breathing is sighing or panting in character; the rate is increased; the movements of inspiration and expiration are of nearly equal length. Expiration lacks the characteristic prolongation of asthma. The chest lacks the typical distension of asthma. In asthma breathing is spasmodic, with inspiration short; expiration, prolonged and labored. The rate of respiration not increased.

PERCUSSION.—In *cardiac dyspnœa* the note over the upper portion of the chest is normal. Over lower portion of the chest, especially at the base, posteriorly, dullness is more or less marked, in proportion to the amount of pulmonary œdema and passive congestion.

In asthma the note is hyper-resonant over all portions of the chest, especially at the base.

AUSCULTATION.—In cardiac dyspnœa during inspiration, vesicular murmur is present over upper portion of lung; over base it may be feeble or absent, according to the amount of pulmonary congestion and œdema.

The relative length of inspiration and expiration is but slightly altered. Râles, both dry and moist, may be present, but dry râles are not as varied and are more constant than in asthma; while moist râles are relatively more abundant, especially over the base. In chronic passive congestion (cardiac pneumonia) the breath sounds may have a bronchial quality. When dyspnœa is due to valvular disease, the physical signs of the lesion are usually present. At times the distinguishing signs in the lung may be intense enough to mask the cardiac murmurs.

In asthma dry râles are the most prominent. When detected, breath sound is normal, never bronchial.

Laryngeal and Tracheal Stenosis. INSPECTION.—Dyspnœa is inspiratory with increased movement of the larynx. Size of the chest is diminished; the diaphragm is elevated; there is marked retraction of the suprasternal, supraclavicular and intercostal spaces. Inspiratory portion of respiration is prolonged and labored. Expiratory movement is shorter than normal. In asthma the dyspnœa is chiefly expiratory; the chest is enlarged; the diaphragm is depressed.

PERCUSSION.—In stenosis the percussion note has diminished resonance. In asthma it is hyper-resonant.

AUSCULTATION.—In stenosis, vesicular murmur over the lung is diminished or feeble; the stridor, due to the narrowing of the larynx, frequently cause bronchial type of breathing to be heard over both lungs. The changes in the respiratory sounds are constant. In asthma, dry râles of varying size are heard over a given area, constantly changing in character.

Aneurism of the Aorta.—Compression by the aneurismal sac of the trachea and left bronchus may simulate very closely an asthmatic attack. The physical signs are not bilateral. The intensity of the signs will depend on the degree of narrowing. When the bronchus is only slightly narrowed, the respiratory sound heard over the lung beyond the point of constriction may have a slightly tubular or a marked sonorous quality. Entire occlusion of the bronchus causes absence of breath sound over portion of lung supplied by the tube. Dullness over the sternum and tracheal tugging, brassy cough, and heaving impulse are generally sufficient for diagnosis.

Hysterical Breathing.—Paroxysmal and labored breathing, similar to that of asthma, occurs during hysterical attacks. Expansion of the chest, due to voluntary action of the muscles of inspiration, and voluntary interference with expiration, may be identical with that of asthma. The rate of the breathing is generally increased.

On auscultation the dry râles, with prolonged expiration are not present.

PULMONARY TUBERCULOSIS.

Synonyms.—Phthisis, consumption.

Pulmonary tuberculosis is a disease of the lungs, due to the presence of the tubercle bacilli, with the production of tubercular nodules.

The distribution of the tubercles may be localized or diffuse. The primary effect is the same in both cases, varying merely in degree. When localized, its point of selection is usually the apex, although certain factors may determine its primary location in other portions of the lung. When more widely distributed, it may involve an entire lobe or lung, or may be disseminated throughout the entire bronchial tract.

Effect Upon the Lung.—Immediate lodgment of the tubercle bacilli usually occurs in the terminal bronchioles. With the production of the tubercular nodule, the bronchiole becomes occluded, and the alveolus supplied by the bronchiole becomes the seat of an inflammatory process, with the production of broncho-pneumonia. The peribronchial tissue is also involved. Extension to neighboring lobules occurs through the lymph spaces and vessels into the interstitial tissue, and also through the bronchi. On account of the relation of the lymphatics of the interlobular tissue to the pleura, there is early involvement of the subpleural tissue, with secondary involvement of the pleural sac, which may, however, not be of a tubercular nature.

Secondary Changes.—Dependent upon tubercular infection, three varieties of pathological changes occur: (1) Congestion, inflammation and œdema of the tissues adjacent to the tubercular nodule, producing more or less complete consolidation of the alveoli, with inflammatory products (catarrhal, broncho-pneumonia). (2) Necrosis (caseation, liquefaction, softening) of the infected area. (3) Growth of connective tissue (fibrosis). These three processes occur together, and are present in varying degrees. As one or another predominates in an individual case, the rapidity of its course and the type of the disease is determined.

Two forms of pulmonary tuberculosis occur: A. Acute. B. Chronic. These can be still further subdivided into types, according to *(a)* distribution of the infection; *(b)* the character of the changes in the infected area.

A. Acute Pulmonary Tuberculosis.

Three types may be recognized clinically: (1) Acute miliary tuberculosis, pulmonary type. In this form of the disease the tubercle bacilli are widely distributed throughout the lung. The eruption of tubercles is very rapid, and the inflammatory changes in the alveoli and adjacent bronchi are very marked. These early changes in the lung are almost identical with those that occur in acute capillary bronchitis with involvement of the alveoli. The necrosis and liquefaction occur almost at once, while there is almost complete absence of connective tissue increase.

(2) Broncho-pneumonic type. In this form the eruption of tubercular nodules is at first localized in the apex of one or both of the upper lobes, or tubercular foci may be scattered throughout the lung. The areas involved in the tubercular process are the seat of an intense catarrhal inflammation, and caseation, with liquefaction and cavity formation, occurs very rapidly; so that the lung passes in a short time through the stages of infection, tuberculization, infiltration (broncho-pneumonia), and softening, with the production of a number of small cavities. The tubercular masses may coalesce, forming a confluent broncho-pneumonia.

(3) Lobar type. Occasionally the amount of tissue involved by a confluent area of tubercular broncho-pneumonia may be so great as to involve nearly an entire lobe, giving consolidation as extensive as is found in acute croupous pneumonia.

On account of the rapidity of the changes and the nature of the physical signs, various names have been given to this form of acute pulmonary tuberculosis, as acute phthisis, acute pneumonic phthisis, tubercular pneumonic phthisis, caseous tubercular pneumonia, broncho-pneumonic phthisis, acute catarrhal phthisis, epithelial phthisis, florid phthisis, galloping consumption.

Physical Signs.— In the acute miliary type the earliest changes may be those of general bronchitis, gradually increasing in intensity with the occurrence of broncho-pneumonia. In the early stage of acute miliary tuberculosis, the signs are identical with those described under Capillary Bronchitis with involvement of the bronchioles and alveoli (page 144), the dyspnœa, however, is more marked and persistent. Where the disease is localized, the following signs may be present:

INSPECTION.—In the broncho-pneumonic type, in the early stage, there is no change in the shape of the chest. With occlusion of a large number of bronchi and pulmonary collapse, there is depression of the thorax over the affected area, with loss of motion. With the occurrence of liquefaction (softening) and the formation of cavities, the depression may become less marked, and motion may return.

In the lobar type there may be loss of motion over a wider area, while more complete consolidation of the lung causes absence of thoracic depression.

PALPATION.—In the broncho-pneumonic type vocal fremitus may be normal when the disease is deep seated, and feeble when there is marked pulmonary collapse. When the consolidation is close to the surface, vocal fremitus is slightly increased.

In the lobar type vocal fremitus may be as intense as in true croupous pneumonia.

PERCUSSION.—In the early stage the percussion note may be normal. Over the portion of lung affected by obstruction of the bronchi, the percussion note is dull, with slight tympanitic quality. Later there is gradual increase of dullness, according to the degree of consolidation. When pleural changes are present, dullness may almost reach the degree of flatness. With cavity formation, the dullness is replaced by cavernous or amphoric percussion resonance.

AUSCULTATION.—The earliest signs over the affected area are those of diffuse bronchitis, with sibilant and sonorous râles and moist râles of a liquid, bubbling character. The breath sounds may be weak in occlusion of the bronchioles and pulmonary collapse. As the collapsed lung becomes consolidated by inflammatory products, the breath sounds become bronchial in character, but rarely tubular. The liquid, bubbling râles of bronchitis are replaced by sharp, crepitating râles and by pleuritic friction sounds. As softening occurs, the small râles are replaced by coarser ones, and the bronchial breathing loses its sharpness and has a hollow, blowing quality; it rarely becomes markedly cavernous or amphoric. The slower the course of the disease, the greater the tendency for the areas of consolidation to coalesce, with a corresponding increase in the intensity of the bronchial quality of the breath sounds. As the different tubercular areas do not undergo the changes of consolidation, softening and cavity formation with the same degree of rapidity, the physical signs of all stages of the disease will be found in different portions of the lung. In the localized broncho-pneumonic and lobar types, the disease may at any stage lose its acute character and pass into the subacute or chronic form.

Differential Diagnosis. *Simple Broncho-Pneumonia.*—As stated above, the early physical signs are identical in the tubercular and non-tubercular forms. As the disease advances to the stage of softening, slight differences may be noted. In the simple, non-tubercular pneumonia resolution occurs at a uniform rate over the affected areas, with a gradual return to the normal condition. In the tubercular type, with softening and destruction of the lung tissue, râles become larger than those common in bronchi of normal size. The bronchial character of the breath sounds is gradually replaced by soft, blowing, puffing breath sounds, especially marked on expiration. In the lobar pneumonic form the physical signs may be for a time identical with those of croupous pneumonia, but later certain changes develop in the consolidated lung which differ. The vocal fremitus over the consolidated area, while it may be as intense as that found in the second stage of croupous pneumonia, is not uniform over all portions of the lobe involved. During the period of softening, vocal fremitus does not diminish to the same degree in the tuberculous cases. On auscultation, fine crepitating râles of the first stage of croupous pneumonia may be present, but they are not as fine nor as sharp, and do not occur in well-defined showers. They do not disappear with the occurrence of bronchial breathing, as in croupous pneumonia, but persist until replaced by the coarser râles of softening. With the beginning of softening, the râles of tubercular pneumonia may be identical with those of resolving croupous pneumonia (râles redux), but they tend to become larger, and are later replaced by mucous clicks and gurgles. In croupous pneumonia the râles are uniform in size, and become fewer as resolution occurs.

The bronchial breathing of tubercular pneumonia does not disappear with the signs of softening, but, while it may for a time be more or less masked by râles, it reappears over scattered areas and has a hollow, cavernous or amphoric quality. In croupous pneumonia the bronchial quality of the breath sounds gradually fades out as resolution proceeds.

While the above physical signs may aid in differentiating tubercular from non-tubercular lobar pneumonia, the most important data are obtained by the temperature, duration and course of the disease, and microscopical examination of the sputum. In tubercular pneumonia the physical signs of softening are not associated with the phenomenon of crisis and lowered range of temperature. The expectoration of tubercular pneumonia is apt to be grayish-green (Traube), differing markedly from the tenacious, blood-stained (brick dust) sputum of croupous pneumonia.

(B) Chronic Pulmonary Tuberculosis.

In the subacute and chronic forms of pulmonary tuberculosis, following local infection by the tubercle bacilli, pathological changes occur which are of the same general nature as those of the acute form, but differ in the degree of severity and in their course.

In these forms the disease is more distinctly localized. With the development of the tubercular nodules, there may be congestion, inflammation and œdema of the affected lobules, giving the pathological changes and physical signs of localized acute bronchitis or acute pneumonia, as was described in the acute forms. These acute conditions subside, and the disease runs a milder, more subacute or chronic course. In the large majority of cases the incipient stage is more insidious, and with the local infection there is little or none of the acute inflammatory change, but a progressive increase of the connective tissue (fibrosis), which extends outward from the site of infection along the interlobular septa to the surface of the lung, and early involves the pleural and subpleural surfaces, causing thickening and adhesion. The bronchi in the affected area are narrowed or occluded, with subsequent pulmonary collapse. The fibrosis and changes in the bronchi and alveoli cause imperfect expansion of that portion of the lung and retraction of the tissues of the thorax over it, unless the functionless portion is compensated for by the adjacent overlying lobules. During the early or incipient stage, all degrees and extent of these pathological changes may be present at different times in the same case. Their relative preponderance determines the type of the disease and influence its course. The physical signs present will be those of localized bronchitis, catarrhal pneumonia, fibrosis and pleurisy. The more marked the fibrosis, the more latent and chronic the course.

The disease may be arrested in this stage by encapsulation of the tubercular area by a dense fibrous envelope. Usually (second stage) there is extension of the tuberculous infiltration to adjacent portions of the same lobe, as described below. This extension is along fairly definite lines, and is usually associated with more or less consolidation, due to connective tissue increase. With the extension of the disease there is corresponding involvement of the pleura, which may be acute, subacute, adhesive or pleurisy with effusion. The consolidation is never as complete as in croupous pneumonia.

Sooner or later, in the majority of cases, necrosis (third stage) of the tubercular areas occurs, followed by liquefaction and softening, with the formation of cavities of varying sizes. These later changes may first show themselves in the primary focus, or, when fibrosis has been more marked, condensation may occur without cavity formation; while breaking down occurs in those portions secondarily affected. This tendency of chronic pulmonary tuberculosis to necrose and form cavities has given it the name of *chronic ulcerative tuberculosis.* Necrosis and softening occur especially in those cases in which the inflammatory changes (broncho-pneumonia) have been most marked. Where fibrosis has predominated, the disease may present the changes that were described under chronic interstitial pneumonia (page 174). Of equal importance with the pathological changes and physical signs is the distribution of the primary foci and the manner of extension of the disease throughout the pulmonary system.

"Tubercular disease, in its onward progress through the lung, in the majority of cases follows a distinct route, from which it is only turned aside by the introduction of some disturbing factor." ("Localization of Lesions of Phthisis," Fowler.)

Site and Progress of Pulmonary Tuberculosis.—The most common point of primary infection is at the apex of the upper lobe of the lung, 1 to 1½ inches below the summit. This may be either on the anterior or posterior border. Anteriorly, this point corresponds to the supraclavicular and the infraclavicular space opposite the middle of the clavicle, posteriorly to the supraspinous fossa. From this point of origin, the disease tends to involve the upper lobe, following along the anterior border. Yet not infrequently the primary localization is on the outer portion of the upper lobe, corresponding to the first or second interspaces, just below the outer third of the clavicle—a localization less favorable for arrest or cure than the first. After involvement of one upper lobe, the secondary tubercular deposit may appear at the apex of the opposite lung, but generally there is early involvement of the upper portion of the lower lobe on the same side, at a point which corresponds to the midscapular space opposite the fifth dorsal spine. When the physical signs over the apices of the lung are doubtful, the presence of change in the breath sounds or the occurrence of râles over this position is of great diagnostic value, as it almost invariably indicates that the signs at the apex are due to tubercular changes. The base of the lower lobe is rarely *primarily* affected, so that if the respiratory sounds over the apices are normal much caution must be exercised in pronouncing the changes that occur at the lower portion of the lower lobes tubercular. Secondary foci in the upper portion of the lower lobe occur soon after primary infection of the upper lobe on the same side, generally extending backwards along the posterior surface of the lung and along the line of interlobular septa. The interlobular septa and the area of usual extension correspond to the lower border of the scapular when the arm is raised above the level of the clavicle, and the hand carried well over the shoulder of the opposite side until the finger tips rest upon the spine of the scapula. Extension downward generally occurs by the development of separate foci, and not by gradual extension from the upper portion of the lobe. Primary basic tuberculosis is extremely rare. In estimating the probability of basic lesions being tubular in origin, the following points must be considered:

1. If the physical signs of the disease in the lower lobe are continuous from apex to base, it is probably tuberculosis.

2. If the base is affected, but the upper part of the lower lobe is free from all change, the basic lesion is probably *(a)* non-tubercular; it may be collapse following bronchiectasis, catarrhal pneumonia, pneumonia or pleurisy. *(b)* "If tubular, it is secondary to some condition which has diminished the normal resisting power of the base, *i. e.*, pleurisy followed by partial collapse, for example; but the presumption is strongly in favor of non-tubercular lesion." (Fowler.)

The apex of the opposite lung is generally involved after the lower lobe on the primarily affected side. The lesion has usually the same site as in the lobe primarily affected, but may be close to the interlobular septum, corresponding on the chest wall to the upper part of the axilla. The middle lobe of the right lung is rarely the seat of the primary lesion. It is generally affected rather late by extension from the upper lobe on the same side, and may escape entirely.

Physical Signs.—The physical signs present in subacute and chronic pulmonary tuberculosis during the different stages vary with the nature of the pathological changes, at one time approaching those of the acute type, to again become less acute, closely simulating those of chronic bronchitis, chronic interstitial pneumonia or chronic pleurisy. One of the most distinctive features of the physical signs of chronic pulmonary tuberculosis is their localization for a longer or shorter time, especially at the apex, and their *gradual* extension throughout the lung. This fact allows of division of the signs into those diagnostic of three stages of the disease: (1) The early or incipient stage; (2) the stage of extension and consolidation, and (3) the stage of softening and excavation. These stages cannot be defined with positive sharpness, but they are useful to indicate the nature of the preponderating pathological changes; and the same case may show all stages present in different portions of the lung, or even in one lobe.

INSPECTION. *Early or Incipient Stage.*—The thorax over the affected area may show no change in size or shape, or early there may be depression of the bony thorax. Flattening of the upper portion of the thorax is suggestive of tuberculosis, especially when the flattening is limited to one apex. Frequently it can only be noted when the patient is lying flat, or when looking down over the shoulder.

When fibrosis and pulmonary collapse are the predominating pathological changes, the deformity of the thorax is marked. There is deepening of the supra- and infraclavicular spaces, and retraction of the first and second ribs, and the clavicle becomes somewhat prominent. With these changes in the bony thorax, the expansion over the affected area is markedly diminished, while that of the opposite side is increased. Pleural involvement with pain gives still further diminution of motion. Changes in the bony thorax may be entirely absent when dilatation of the air-vesicles (compensatory emphysema) occurs around the affected area.

Second Stage.—As the disease advances, the signs noted by inspection are influenced by the amount of fibrous induration present. When this is slight, the chest may show very little deformity, although motion of the thorax may be markedly diminished. When fibrous thickening of the lung is marked, there is localized flattening of the chest and marked loss of expansion. Extension of the disease to the upper portion of the lower lobe causes the scapula on the affected side to become more prominent, while at the same time there may be curvature of the spine.

Third Stage.—With the occurrence of softening when the pneumonic changes have been the most marked feature, with a slight amount of fibrosis, the deformity of the previous stage may be lessened, while at the same time expansion over the affected area becomes freer. Frequently, when the apex of one lung is in the third stage, with early secondary involvement of the opposite side, the expansion will be freer on the side first affected.

In some cases marked softening with cavity formation does not occur, being replaced by interstitial changes in the affected area. In these cases there is marked contraction of the chest, producing curvature of the spine. When situated in the upper portion, there is depression of the shoulder, with projection of the scapula. The intercostal spaces are depressed, and the ribs may overlap each other; expansion is diminished or absent. When the upper lobe of the left side is affected, the apex beat of the heart is displaced upward and slightly outward, and may be seen in the third left interspace, or even higher. When the lower lobe of the left side is involved, the apex beat may be seen in the left axilla. Fibrosis of the right upper lobe causes the heart to be displaced toward the right, the apex beat frequently being invisible, because it is under the sternum. Contraction of the right lower lobe causes the apex beat to be seen at times beyond the right border of the sternum.

PALPATION *Early or Incipient Stage.*—It must be borne in mind that vocal fremitus beneath the right clavicle is greater than that on the left, and serves as a standard for comparison. If vocal fremitus is equally well marked on both sides, it indicates that there is either diminution on the right side or increase on the left.

In the early stage vocal fremitus may be normal, increased or diminished over the affected area. It is normal when the lesion is deep seated and covered with normal lung; increased when there is inflammatory change or marked fibroid thickening; diminished when pulmonary collapse or pleuritic thickening is marked. Change in vocal fremitus in tuberculosis is to be taken in connection with the other physical signs. Palpation is frequently more accurate than inspection in showing slight change in the expansion over the apices.

Second Stage.—With extension of the disease beyond the primary foci, vocal fremitus may show the same variations as noted during the first stage. Usually extension is accompanied by well-marked inflammatory changes in that portion of the lung that is secondarily affected, and there is a corresponding increase in vocal fremitus. Involvement of the upper portion of the lower lobe causes increased vocal fremitus over the interscapular space. Further extension over the lower lobe causes increased vocal fremitus in parts which are not contiguous. Involvement of the opposite side of the upper lobe may show the same changes as those that occur in the primary foci.

Third Stage.—Vocal fremitus may be increased with the beginning of softening, due to the occurrence of acute exacerbations, which frequently accompany this change. When cavities are formed, vocal fremitus becomes less marked. When fibroid changes predominate in the later stages, it may be increased. Thickening of the pleura frequently causes diminution of vocal fremitus.

PERCUSSION. *First Stage.*—The percussion note may be unchanged, but the earliest departure from normal is usually a slight rise of pitch, with diminution of resonance. Percussion of the apex is most important. Percussion should be uniform and not too forcible, and the tissues of the thorax should be lax. The height to which the apex of the lung rises above the clavicle during inspiration and expiration should be determined, as diminished expansion on one or both sides is frequently the earliest symptom. Posteriorly, dullness on percussion, with marked heightening of the pitch, may be noticed in the supraspinous region. Thickening of the pleura causes the note to be dull and higher pitched, with increased sense of resistance.

Second Stage.—As consolidation extends, dullness will be detected along the edge of the sternum, and extending toward the axillary line. Careful percussion should be made at the apex and in the axillæ in all cases. Involvement of the upper portion of the lower lobe causes marked dullness in the interscapular space. When one apex is affected, careful examination for change in pulmonary resonance should be made frequently over the opposite apex.

Third Stage.—Softening in the affected area may early cause a slight return of resonance, with tympanitic quality over the area where the percussion was dull and high pitched during the stage of consolidation. With destruction of lung tissue and the formation of a cavity, the percussion note becomes cavernous or amphoric, according to the condition of the walls of the cavity. Marked pleuritic thickening with fibroid change causes the note to become dull, with a well-marked boardy or wooden quality.

AUSCULTATION. *Early or Incipient Stage.*—Usually the earliest signs are the occurrence at the apex of fine, moist râles or crepitations, which are noticed during inspiration. These may be detected only after coughing, and may be limited to one or two râles. An important feature of these râles is their persistence at the point where first heard. Dry râles over one apex at times precede even moist crepitations, and are associated with feeble inspiratory breath sounds. These auscultatory signs generally antedate the early signs noted by inspection, palpation and percussion.

The respiratory murmur may be affected in various ways during the incipient stage. It may be *(a)* feeble, wavy or jerky in inspiration or expiration, or in both. This change is generally associated with occlusion of the bronchioles and collapse of the air-vesicles. *(b)* Wavy inspiration, with prolonged, high-pitched, slightly blowing expiration. This occurs when there is more marked increase in fibrous tissue. *(c)* The inspiratory breath sounds are harsher, higher pitched, with slightly tubular quality, while the expiratory is prolonged so that its length equals or exceeds that of inspiration; its pitch is higher and its quality blowing or tubular when the inflammatory changes are most marked (bronchopneumonia).

It is necessary to call attention again to the normal breath sounds that may occur over the right apex. Normally, these are slightly harsher than on the left side, and in certain anatomical arrangements the bronchi themselves may have a well-marked tubular quality. The relative intensity of the breath sounds on the two sides has the same diagnostic importance as the vocal fremitus.

Second Stage.—As the disease advances, the auscultatory signs change, according to (1) the extent of consolidation, giving the more intense type of bronchial breathing; (2) whether or not there is accompanying bronchitis when dry and moist râles are present; (3) fibrosis and induration, causing marked increase in the length of expiration; the bronchial element is not as marked, but is higher pitched than in the bronchopneumonic type. (4) Thickening of the pleura causes various types of friction râles to be heard, with diminution or absence of the breath sounds. (5) Compensatory distension of the lobules surrounding the affected area sufficient to cause the signs of consolidation to be masked by those of compensatory emphysema.

Secondary involvement of the upper portion of the lower lobe is shown by fine râles and pleuritic crepitations, with tubular breathing in the interscapular space and over the hilus of the lung. The character of the breathing heard at this point is peculiar, on account of the relation of the upper portion of the lower lobe to the primary bronchi.

Third Stage.—The occurrence of moist râles and their change in size when bronchitis has been a prominent symptom are the first evidence of softening. At first these râles have, in addition to their liquid quality, one of sharpness, dependent upon the consolidated condition of the lung in which they are made. But the conversion of the consolidated area into a cavity causes the breath sounds of the second stage to be changed in character. The pitch is lower, and the well-marked tubular quality is replaced by one of more hollow character, cavernous or amphoric. If the cavities are dry, no râles may be present. If they contain fluid râles of large size, gurgles will be present.

Vocal resonance over the area of softening changes in intensity, the broncophony and pectoriloquy of the stage of consolidation being replaced by amphoric or cavernous voice sounds. The stage of softening is rarely uniform throughout different portions of the lung, except in the more acute forms of the disease.

ACTINOMYCOSIS.

Infection of the lung by the ray fungus occurs generally secondary to that of the mouth or other portions of the respiratory tract. Occasionally it may occur primarily in the lung. Clinically, two stages are recognized.

First Stage.—This is characterized by gradual involvement of the lung and pleura. During this stage the physical signs are those of tuberculosis of the lung or emphysema, according as the pulmonary or pleural element is predominant. Usually the primary focus is at the base of the lung.

INSPECTION.—Restricted motion over the lower portion of the affected side. With extensive involvement of the lung or pleura, there is increased bulging.

PALPATION.—As there is more or less occlusion of the bronchi in the affected area, vocal fremitus is diminished. Extension to the pleura is attended with absence of vocal fremitus.

PERCUSSION.—Flatness, with increased sense of resistance.

AUSCULTATION.—Feeble or absent respiratory murmur and pleuritic frictions.

Second Stage. INSPECTION.—This is characterized by extension of the growth to the chest wall, and the involvement of the various structures. There is absence of motion over the affected area. The intercostal spaces are filled. Infiltration of the soft parts causes localized inflammatory signs. With necrosis of affected tissue, there is discharge of characteristic pus.

PALPATION.—Over the intercostal tissue there is a sense of hardness in the interspaces and a brawny, œdematous condition of the soft parts externally. With breaking down, before free opening occurs, fluctuation may be detected.

PERCUSSION.—Before the opening occurs on the surface of the chest, the signs may be that of localized empyema. The percussion outline does not follow that of effusion into the free pleural cavity, although it may be identical with that of localized empyema. The diagnosis can frequently only be made by puncture and microscopical examination of the fluid obtained.

SYPHILIS OF THE LUNG.

Syphilitic lesions of the lung may occur as (1) gumma, (2) broncho-pneumonia, (3) interstitial pneumonia (syphilitic phthisis), (4) involvement of the bronchial glands and lymphatics. Destructive disease of the lung due to syphilis is very rare.

The physical signs that accompany the different changes in the lung are closely allied to those dependent upon tubercular infection, and are not distinctive. Diagnosis depends largely upon the distribution of the foci and the subsequent course.

Tubercular Disease.	*Syphilitic Disease.*
Pulmonary tuberculosis primarily involves the apex, and its extension throughout the lung follows a definite line.	Syphilis usually affects the hilus and central portion of the lung.
Coincident with the extension of the disease, softening and cavity formation occur.	With extension softening and destruction of lung tissue rarely occur.
When cavities occur they tend to enlarge with concurrent signs of softening and consolidation.	Cavities, when present are of the bronchiectatic type, and are attended with the signs described under bronchiectasis.
Fibrosis in pulmonary tuberculosis is usually attended with secondary changes of consolidation and softening.	Fibrosis excessive, distribution through the lung follows the line of lymphatics in the interlobular spaces.
Stenosis of the bronchi rare.	Stenosis of bronchi and larynx commonly associated with change in the glandular structures at the hilus.

In many cases the differential diagnosis can be based only on repeated examinations of the sputum and the absence of tubercle bacilli, especially when signs of softening and bronchitis are present.

MALIGNANT DISEASE OF THE LUNG.

Primary involvement of the lung is rare. It is usually dependent upon extension from the mediastinal and adjacent structures or metastasis from distant portions of the body. It is especially liable to occur after operation on the mammary gland.

Physical Signs.—These are variable, according to the site of the disease and whether the pleura is involved.

INSPECTION.—When deep seated or localized, inspection may be negative. With occlusion of a bronchus, localized retraction of the chest may be present. Extension to the pleura causes marked local bulging. When the entire pleural sac is involved, there is excessive enlargement of the affected side, exceeding that of effusions into the pleural cavity.

PALPATION.—Vocal fremitus is increased unless the growth occludes a bronchus or involves the pleural cavity, when it is diminished. The cardiac impulse may be displaced, and pulsations may be transmitted through the new growth to abnormal areas.

PERCUSSION.—Change in the percussion note varies according to the situation of the growth and the character of the surrounding tissue.

AUSCULTATION.—The sounds are those of consolidation, pulmonary collapse or thickening of the pleural sac. When extension to the lung is secondary to mediastinal involvement, pressure symptoms are present.

PLEURISY.

Pleurisy is an inflammation of the serous membrane, characterized by an exudation upon the free surface of the pleura, which may be fibrinous, serous, sero-fibrinous, sero-purulent, purulent or hemorrhagic. All these varieties of exudation may occur at different stages of the same case of pleurisy. The inflammation may not be attended with an exudation upon the free surface, but characterized by a fibrous thickening. Inflammations of the pleura may be acute or chronic, general or localized, primary or secondary. Numerous classifications or subdivisions of pleurisy have been made. For physical diagnosis three forms are sufficient, depending upon the character and amount of the exudation and the changes produced in the pleura: (1) Dry pleurisy with fibrinous exudate; (2) pleurisy with effusion, and (3) chronic pleurisy with adhesions.

(1) **Dry Pleurisy.**—Before the exudation appears the pleural surface undergoes certain changes, which may give quite distinctive physical signs. The normal

moist condition of the pleural surface is lacking. The surfaces becomes dry; there is loss of lining endothelium, with more or less roughening of the pleural sac. When exudation takes place, the pleural surfaces are more or less thickly coated with fibrinous material, which, while allowing a certain amount of movement between the two pleural surfaces, causes thickening of the pleural sac and the occurrence of physical signs.

(2) **Pleurisy with Effusion.**—This form includes serofibrinous pleurisy, subacute pleurisy, chronic pleurisy with effusion, empyema. The nature of the fluid does not modify the physical signs. The physical signs that are present with effusion into the pleural cavity are dependent upon the presence of the fluid and the effects that it produces upon the lung, heart, position of the diaphragm and abdominal viscera and in the thoracic walls. Normally the pleural surfaces are in apposition throughout. They are kept together by atmospheric pressure, although the elasticity of the lung on the one side and that of the thorax on the other produces a constant traction on the two pleural surfaces, which tends to separate them. When fluid or air is present in the pleural cavity, certain changes occur: *(a)* The two surfaces of the pleura are separated; *(b)* the distension of the pleural sac diminishes the space to be filled by the lung; *(c)* the elasticity of the lung causes it to retract and to exert traction or lifting power on the fluid. Until a certain point is reached, the fluid exercises no compressing action on the lung, which retracts before the fluid until its elasticity is satisfied. After this point has been reached, the fluid causes compression of the lung. *(d)* With the filling in of the pleura there is loss of tension in the pulmonary tissue, producing change in the percussion note. *(e)* The chest walls, relieved from the pulling action of the lung, assume the position of elastic equilibrium, changing the contour of that side of the thorax. The diaphragm, unacted upon by the lung, does not rise as high in the thorax; later on the

weight of the fluid causes it to descend still lower. *(f)* The opposite lung is also influenced by the change in the interthoracic pressure, and tends to contract and draw the heart and mediastinum towards the unaffected side. Gradually, with the accumulation of fluid, the normal interthoracic negative pressure diminishes to zero, and later may be replaced by positive pressure. As long as a negative pressure is present in the chest, it exercises an action upon the fluid. As the fluid increases the negative pressure becomes less marked, and later the weight of the liquid itself exerts a positive pressure, causing bulging of the intercostal spaces, compression of the lung, depression of the diaphragm, displacement of the heart and interference with return circulation. As the negative pressure depends upon the elasticity of the lung, the occurrence of signs indicating change from negative to positive pressure is an index of the elasticity of the lung when taken in connection with amount of expansion.

(3) **Chronic Pleurisy with Adhesion.**—In chronic pleurisy with adhesion, more or less fluid may be present, but usually the most marked pathological change is fibrous thickening of the pleura with adhesions, binding together the two surfaces of the sac. The effect of thickening of the pleura is to interfere with expansion of the lung and to prevent free movement of the thorax. When these changes are localized, their effects are limited to the area involved, causing many of the thoracic deformities and interference with respiratory movements noted under "Inspection" and "Palpation." Thickening of the pleura, with imperfect expansion of the pulmonary tissue, causes change in the percussion note. As explained under "Auscultation," imperfect expansion of the lung causes diminution or absence of the normal breath sounds. Localized adhesive pleurisy occurs early in pulmonary tuberculosis, and modifies the physical signs of that condition.

Physical Signs. INSPECTION. *1. Dry Pleurisy.*—

When pain is present, the patient leans to the affected side, or lies on that side to prevent motion. Expansion is restricted, and motion is short and jerky. Movement over opposite lung is exaggerated.

2. Pleurisy with Effusion.—When effusion, either fibrinous or serous, is sufficient to separate the two surfaces, with absence of pain, motion over the affected side may be freer. With the occurrence of liquid effusion, there is slight enlargement of the lower portion of the thorax. As effusion increases, the contour of the side becomes more rounded, with widening of the intercostal spaces. With complete filling of the pleural sac, the side is enlarged in all directions. The spine is slightly curved toward the affected side; the intercostal spaces are obliterated, and the chest appears smooth. Motion becomes progressively less until, with full distension, it is absent. When positive pressure occurs within the pleural sac, there is bulging of the intercostal spaces, and pulsations may be present. The opposite side is in a state of compensatory emphysema, and the respiratory movements are exaggerated. Cardiac displacement occurs early unless prevented by adhesions or conditions causing fixation of the mediastinum. With absorption of the fluid or with its withdrawal, the chest returns gradually to its normal contour, with increase of the respiratory movements.

3. Pleurisy with Adhesion.—The occurrence of pleural thickening or adhesions causes movement to be restricted, followed by retraction of chest walls on the affected side if pulmonary expansion is prevented. These changes are especially marked over the base of the lung.

PALPATION. *1. Dry Pleurisy.*—Vocal fremitus may be normal. When the fibrinous exudate is thick, vocal fremitus may be diminished or absent. Slight friction fremitus may be detected.

2. Pleurisy with Effusion.—With the occurrence of effusion, there is gradual loss of vocal fremitus, occurring first in the lower portion of the pleural sac posteriorly, and gradually advancing upward as the sac becomes filled. When the fluid has reached a certain amount, vocal fremitus is usually increased at the upper level. Vocal fremitus over the upper lobe of the lung may be normal. With extensive effusion, relaxation of the lung causes absence of vocal fremitus over the upper lobe also. Compression of the lung causes increased vocal fremitus posteriorly along the spine. Vocal fremitus may be detected over the site of the fluid when adhesions bind the lung to the chest wall and prevent its retraction. The degree of cardiac displacement may be estimated more definitely by palpation than by inspection. Displacement of the liver below the free border of the ribs occurs when the effusion is large, or when retraction of the lung or loss of elasticity cause the fluid to act upon the diaphragm early.

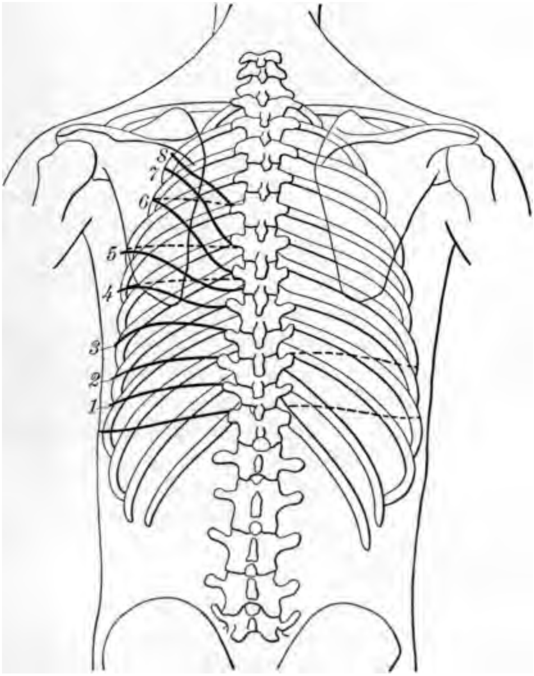
3. Pleurisy with Adhesion.—Pleuritic thickening, besides interfering with respiratory movements, causes diminution of vocal fremitus; and these, when associated with fibrous induration of the lung, may cause increased vocal fremitus.

PERCUSSION. *1. Dry Pleurisy.*—With a slight fibrinous layer, there is little or no change in resonance. If exudation is very abundant, there is loss of resonance, with increased sense of resistance.

2. Pleurisy with Effusion.—With the occurrence of effusion, the earliest indications are shown by flatness at the base of the lung posteriorly. Slight effusion causes the area of flatness to be widest along the spine, sloping in a gentle curve toward the axilla. With increase of fluid, the line of dullness extends beyond the axilla to the mammary line. When the fluid is sufficient to reach the angle of the scapula, the percussion outline shows a more or less curved line, which has been called the "curved line of Ellis."

The fluid rises highest in the axilla, and on the anterior border of the chest the line curves slightly downward toward the sternum, while posteriorly the line descends more abruptly to the spine. Percussion of the affected side at this stage shows three zones: (1) Flatness over the fluid; (2) posteriorly, dullness over the triangular space, bounded above by a line drawn horizontally from the apex of the line of flatness in the axilla. Above the horizontal line pulmonary resonance of low pitch, with faint tympanitic quality, may be detected. (3) On the anterior portion of the chest,

FIG. 27.

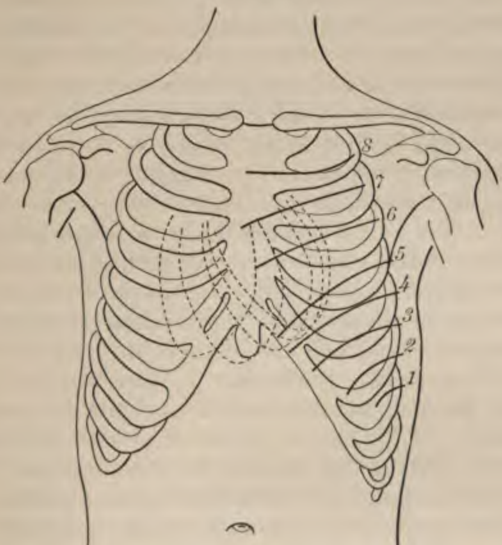


Lines 1, 2, 3, curve of small effusion.
" 4, 5, 6, 7, curved line of Ellis (letter of S).
Dotted line from summit of curve to spine, upper limit of dull triangle.

between the horizontal line and the line of flatness, percussion gives a peculiar tympanitic quality—Skoda's resonance. (Figs. 27 and 28.)

As the fluid increases in amount and the elasticity of the lung is satisfied, the fluid assumes a more horizontal line, and at last, when negative pressure has been replaced by positive (which generally occurs when the fluid reaches the level of the third rib), the lung is compressed by the fluid, and a small area of tympanitic percussion may be detected anteriorly under the clavicles, close to the sternum, and in the supraspinous fossa behind.

FIG. 28.



Dotted line showing position of heart, according to amount of effusion.

As the curved outline of the fluid depends upon the elasticity of the lung, a more nearly horizontal line, with a small amount of effusion, shows that there is loss of pulmonary elasticity or that the lung is prevented from retraction by adhesions, or that there is consolidation (probably tubercular) at the apex. The position of the line of fluid does not change readily with change in posture of the patient. When the patient has long occupied the recumbent posture, the line of Ellis is more or less displaced by the fluid gravitating toward the most dependent portion.

3. *Pleurisy with Adhesion.*—Fibrous thickening of the pleura with adhesions causes the percussion note to be impaired over the lower portion of the lung, and is usually associated with retraction of the bony thorax.

AUSCULTATION. 1. *Dry Pleurisy.*—The respiratory murmur is jerky and partly suppressed; inspiration is shortened, with expiration prolonged, slow and feeble. During the early stage the dry, rubbing friction sounds, similar to the sound made when two rough surfaces of paper are rubbed together, or when the hand is rubbed over the ear, are heard. With the occurrence of plastic exudation, the character of the friction sounds change. They become stickier, are superficial, and later may have a fine, grazing character. They are most abundant at the end of inspiration and the beginning of expiration. They may be absent in quiet breathing, and only be detected at the end of a deep inspiration. When the pleurisy of the left side involves the portion overlying the præcordia, friction râles may be heard synchronously with the heart sounds.

2. *Pleurisy with Effusion.*—The auscultatory signs are variable. The signs typical of effusion of small amount are slight diminution or absence of breath sounds at base of lung and distant voice sounds. As the fluid increases in amount, there is absence of breath sounds over the lower portion of the thorax corresponding to the line of percussion flatness. At the level of the fluid vesicular murmur may be faintly heard, associated with bronchial breathing of a peculiar blowing,

wavy character. The voice sounds are also of a wavy, nasal character (ægophony). At the upper portion of the thorax, over the apex of the lung, vesicular murmur may be normal or slightly exaggerated. In large effusions at the upper level, bronchial breathing becomes more intense, and bronchophony and pectoriloquy are present.

Occasionally tubular or bronchial breathing, bronchophony and pectoriloquy are heard over the fluid, simulating the auscultatory signs heard in pneumonia. These signs are especially apt to occur in children; also when bands of adhesion prevent the lung from retracting as the fluid accumulates, so that it is compressed posteriorly along the spine. The sound is conducted bv the tense bands of adhesion through the fluid to the bony thorax. Small consolidation of the lower lobes of the lung may also cause bronchial breathing to be present below the level of the fluid.

In addition to displacement of the heart, a soft, systolic murmur is detected over the præcordial space when the effusion is sufficiently large to cause pressure upon the mediastinum or slight distortion of the large vessels. With disappearance of the fluid, the breath sounds return, and pleuritic frictions are heard above the level of the fluid.

3. Pleurisy with Adhesion.—Pleuritic thickening with adhesion causes absence or diminution of the breath sounds, with friction fremitus of a rough, rasping or creaking character. Intercurrent attacks of acute pleurisy may reinduce the signs of fibrinous exudation and of effusion.

Differential Diagnosis. *Pleurodynia.*—Inspection and palpation may be identical. Percussion shows no change in resonance. On auscultation there are present feeble breath sounds, due to loss of expansion of the thorax, but unattended with friction râles.

Hydatid of the Pleura.—This may begin at the base of the lung, but rarely gives a line of dullness so symmetrical as that of pleurisy with effusion. Usually the area of percussion flatness is more nearly circular, with resonance on either side, which may have a more or less tympanitic quality. Displacement of the heart and abdominal viscera does not occur unless the cyst is large. Over the cyst localized bulging of the chest wall is very marked. The differential diagnosis by physical signs between encapsulated pleurisy and hydatid cyst is almost impossible.

Pneumonia. (See page 172.)

Sub-Diaphragmatic Abscess. INSPECTION.—Change in the size and contour of the chest is most marked over the free ribs extending to the hypochondrium.

PALPATION.—Absence of vocal fremitus over lower portion of thorax, associated with rigidity of hypochondriac region and marked depression of liver.

PERCUSSION.—Upper line of flatness is high in axilla, but does not conform to that of pleurisy with effusion at the same level. Absence of dull area behind. Skoda's resonance may be detected anteriorly. Displacement of the heart does not occur. Slight displacement upward of the diaphragm. The apex of the diaphragm is higher than normal. On examination with the X ray the difference between subdiaphragmatic and supradiaphragmatic conditions is well marked.

HYDROTHORAX.

This condition differs from pleurisy with effusion in that it is not inflammatory, but due to simple dropsical effusion into the pleural cavity. When limited to one side, the physical signs are identical with those of the inflammatory type. Usually both sides are involved, and the following physical signs are present:

INSPECTION.—Slight motion on both sides. Slight enlargement over lower portion of thorax.

PALPATION.—Vocal fremitus is most marked posteriorly. Heart is displaced upward.

Percussion.—Flatness over lower portion of thorax. Line of flatness does not follow markedly the curve noted under unilateral affections. Above the level of the fluid Skoda's resonance is early present, with hyper-resonance at the apex.

Auscultation.—Absence of breath sounds at the base, below the level of the fluid. Bronchial breathing at the upper level. Fine, moist râles are heard in the rest of the lungs if pulmonary œdema is present.

PNEUMOTHORAX. HYDRO-PNEUMOTHORAX.

Due to the presence of air or air and fluid in the pleural cavity.

Physical Signs. Inspection. — Enlargement more marked than in pleurisy with effusion. Loss of motion complete.

Palpation.—Absence of vocal fremitus. In hydro- or pyo-pneumothorax succussion fremitus (splashing sounds) is present when the patient is shaken or on violent coughing.

Percussion.—Pneumothorax; increased resonance over affected side. When fluid and air are present in the pleural cavity, two zones of resonance are detected—flatness below the level of the fluid, and increased resonance with a tympanitic quality above it. The line of flatness is horizontal. When the anterior portion of the chest is percussed with two coins while the observer listens behind, a peculiar ringing, metallic sound is heard (coin test).

Auscultation.—In pneumothorax there may be absence of all respiratory sounds over the affected side. When an opening exists between the bronchial tract and pleural cavity, a peculiar, intense, amphoric breathing is heard (lung fistula sound). When fluid and air are both contained in the pleural cavity, by shaking the patient, or after coughing, succussion sounds are promotion as seen at the extremity of the pin or paper cone, and to determine whether or not the movement is expansile. This latter condition is best seen by placing over the area a piece of rubber plaster with a slit cut through it; this will show increase in size by widening of the opening.

Data obtained by inspection must be verified and corrected by the knowledge obtained by the other methods of examination.

Normally, that portion of the thorax which is known as the præcordia shows a slight flattening surrounded by a slight elevation of the ribs.

In the fifth intercostal space, 2½ to 3 inches to the left of the sternum, at a point where a line drawn 2 inches from the left edge of the sternum and 1 inch inside the nipple line bisects the space, is seen a localized, rhythmical impulse occupying an area of about 1 inch square.

THE APEX BEAT.

This is due to the impact of the heart near the anatomical apex on the chest wall. It does not mark, as is commonly supposed, the true anatomical apex, but that part which strikes the chest wall is distant from the true apex about three-quarters of an inch.

The cardiac impulse is due to change in form of the heart during systole, the vertical diameter being lengthened, with a corresponding shortening of the transverse.

The site of the apex beat varies normally within certain limits. In the child it is higher, being found in the fourth interspace, and farther from the mediastinum. In old age it is lower down, being in the sixth interspace, nearer the median line. In persons with short, broad thoraces it is found slightly above the fifth interspace, while in those in whom the thorax is narrow it may be found underneath the sixth rib. and even at the upper border of the sixth interspace. The site of the apex beat also varies with the position of the patient. When lying on the left side, it is carried to the left as far as the midclavicular line, and even further; when the patient is lying on the right side, it is carried inward toward the sternum, but its mobility is not as much as when the patient is lying on the left side. The respiratory act also causes a slight change in the location of the apex beat. In quiet respirations it is but little changed. In forced inspiration it is carried downward and toward the median line, while in forced expiration it is displaced upward and slightly to the left.

Changes in the Præcordia.—The bony thorax over the cardiac area may bulge from the third to the seventh rib, and from the sternum to the left nipple; or the projection may extend from nipple to nipple. The bulging and distortion of the præcordial area may be caused by conditions not associated with cardiac or circulatory diseases: *(a)* Swelling of the cellular tissue, due to inflammatory exudation or to localized deposits of adipose tissue; *(b)* changes in the bony thorax dependent upon rickets; *(c)* a bulging anteriorly, with a compensating posterior curve from spinal curvature.

Local bulgings over the præcordium may be caused by cardiac changes, although they are not marked unless the cardiac disease occurred in early life, when the flexibility of the bones permitted easy distortion. *(a)* Cardiac hypertrophy and dilatation do not produce marked change in the præcordial area unless it has occurred early in life, when it may produce a distinct forward bulging of the sternum and ribs, especially of the left side. Congenital cardiac disease produces the most marked distortion of the thorax. The amount of deformity in the case of chronic hypertrophy with dilatation is important in estimating the time of life at which the cardiac disease occurred. *(b)* Pericarditis causes the contour of the præcordium to be rounded, the normal slight flatness being absent. The interspaces are wider than normal, being even with the surface of the ribs or bulging, according to the amount of exudation into the pericardial sac. The amount of bulging that is present in the case of pericarditis with effusion depends upon the flexibility of the bony thorax; it is marked in childhood, and scarcely noticeable or absent when ossification of the ribs has taken place.

Aneurism at different portions of the thoracic artery may produce local bulgings, as will be described later.

Permanent retraction of the bony thorax does not occur as the result of cardiac disease unless associated with other changes in the thoracic viscera.

Conditions Modifying the Location, Extent and Character of the Apex Beat. — As has already been stated, the visible apex beat merely marks the point at which some portion of the heart near the anatomical apex strikes the chest wall. The portion of the heart which gives the impulse is generally the right ventricle, about three-quarters of an inch from the true apex. The point of contact of the heart may vary within wide limits, due to changes in the relation of the heart to the chest wall, so that the visible apex beat is changed from the normal in location, extent and character.

Influence of the Soft Parts.—The apex beat is much more noticeable and the exact point of contact more distinct in thin persons, especially those with flexible thoraces. In persons where the præcordia is thickly covered with adipose tissue the area of impulse will be slightly increased, while the point of the apex beat will be less distinct. This is especially marked in cases where the mamma is full and tense, the cardiac impulse being conveyed to the entire breast and the apex beat being masked. Where the mamma is large and flaccid, the apex beat may be entirely absent.

Influence of the Bony Thorax.—The elasticity and mobility of the ribs influence largely the character of the apex beat. In children, where there is marked depression of the thorax during expiration, the apex beat is distinct and more extensive. In old age the rigidity of the thorax and secondary changes in the lung cause the apex to become indistinct or absent. Narrow intercostal spaces may render the apex beat invisible, the heart striking the chest immediately under the rib. In very broad thoraces the apex beat is higher, being underneath the fifth rib, or even in the fourth interspace. In long, narrow thoraces the apex beat is lower, being displaced downward and slightly outward. The flat, rachitic or paralytic thorax, with narrow antero-posterior diameter at the lower portion of the præcordium, brings the apex beat further to the left in the fifth interspace. The pigeon-shaped thorax, with increase in the antero-posterior diameter, causes the apex beat to be nearer the median line and somewhat indistinct, except where the depression beyond the bulging causes the true apex to strike the chest wall.

When the ribs over the cardiac region are displaced by primary disease of the lungs, the apex beat is displaced to the right or left and upwards or downward, according to the pulmonary condition.

Influence of Diseases of the Pleura.—Filling of the pleural sac with air or fluid causes displacement of the heart to the opposite side and alters its relation to the chest wall, unless the movement of the heart is prevented by adhesions.

Effusions into the right pleural sac cause the heart to be displaced to the left and upward, the apex beat being seen in the mid-axillary line. The base of the heart is also displaced, but not to the same degree as the apex.

Effusions into the left pleural sac move the heart toward the right, so that, according to the amount of fluid present, the apex beat may be felt at any point between the normal site and the right mammillary line. As was noted under "Pleurisy with Effusion" (page 215), displacement of the heart is one of the most characteristic signs of this condition. Occasionally the cardiac impulse is transmitted to the fluid, giving the physical signs of pulsating empyema.

Adhesions of the pleural surface and contractions draw the heart toward the affected side, with displacement of the apex beat. This will be fully explained later on.

Influence of Changes in the Lung. — Quiet breathing produces no change in the apex beat. Full, deep inspiration causes the inflated lung to cover the heart to a greater degree, thus lessening the extent and force of the apex beat, and may cause it to disappear entirely. Frequently it is sufficient to displace it downward, so that it is seen an interspace lower.

Permanent increase in size of the lungs, as in general emphysema, causes marked change in the relation of the heart to the bony thorax. As a result of the increase in size of the lungs and loss of tension, as was explained under "Emphysema," there is elevation of the ribs and depression of the diaphragm, which also depresses the heart, the apex beat being carried inward and downward. When increase in size of the lung is associated with obstruction of the circulation, the hypertrophy and dilatation of the right ventricle increase the pulsation in area and extent, and carry it still lower, giving epigastric pulsation.

Unilateral emphysema causes the heart to be displaced similarly to the manner seen in pleurisy. Compensatory emphysema in the right lung carries the apex beat to the left and higher. Emphysema of the left lung causes it to be lower, and rarely displaces it beyond the left sternal line.

Shrinkage of the lung as a result of pleurisy, or due to interstitial change in the pulmonary tissue (as in interstitial pneumonia and fibroid phthisis), draws the heart toward the diseased side. When the changes are limited to the lower border of the left lung, that portion of the heart which is normally covered by pulmonary tissue may be brought into direct contact with the chest wall; the visible apex beat will be increased in area and carried horizontally to the left, and a greater portion of the heart, including the true apex, strikes the chest wall. When the change is more extensive, the heart may be drawn upward and outward, the apex beat being seen in the mid-axillary line. When the right lung is affected, the heart is drawn to the right side and upward. Involvement of the upper portion of the lung may cause visible pulsations of the pulmonary artery in the second left interspace, or it may be so extensive that the phases of cardiac movement, systole, diastole and the pre-systolic contraction of the auricle may be seen over the entire præcordial space, giving the impression of a peristaltic wave.

Influence of Changes in the Pericardium.—Effusion into the pericardium causes change in the site and area of the apex beat, according to the character and amount of exudation. In small effusions the apex beat may be seen over a wider area, especially toward the epigastrium. When the pericardium is filled to a greater degree, the visible apex beat is carried upward into the fourth interspace or higher, due to a portion of the heart higher up striking the chest wall, and not to an upward displacement of the true apex. When the pericardium is fully distended with fluid, the apex beat may be absent.

Adhesions of the pericardium to the pleura or mediastinum may cause retraction of the chest wall over the apex (Broadbent's sign). Retraction of the chest wall over the præcordial area is one of the diagnostic signs of adhesions of the pericardium when it produces marked visible recession of the costal cartilages, ribs and sternum, and is associated with signs of cardiac hypertrophy greater than the amount of cardiac valvular disease would produce, and where there is also evidence of a co-existing pleurisy or exo-cardial disease.

Retraction of the chest wall over the apex also occurs with adherent pericardium, secondary to pulmonary tuberculosis.

Influence of Changes in the Heart.—The site, extent and character of the apex beat corresponds, within certain limits, to the extent of the cardiac changes. Powerful action of the heart, due to physical exertion or mental excitement, as in Grave's disease, in poisoning and in fever, causes the apex beat to be more noticeable and broader by causing a larger area of the chest wall to be affected.

In a similar manner, simple hypertrophy of the left ventricle causes the apex beat to be carried to the left and very slightly downward. Increase in the size and force of the left ventricle causes a portion of the heart closer to the true apex to strike the chest wall, and the beat is strong, distinct and defined. When the left ventricle is both dilated and hypertrophied, the apex beat is carried downward and to the left, being seen in the eighth interspace or lower. The impulse is in proportion to the amount of hypertrophy; the apex beat will be correspondingly strong, a large area of the chest wall will be influenced and the impulse, though wide, will be well defined.

When dilatation is the most marked feature, the apex beat will be weak and diffused.

Right-sided cardiac hypertrophy causes the visible pulsation to appear between the ensiform cartilage and the normal position of the apex beat in the fifth interspace. The apex beat may be carried slightly to the left, but rarely goes beyond the left mammary line. When the hypertrophy of the right ventricle is extensive and associated with dilatation, pulsation may be seen in the third, fourth, fifth, sixth and even seventh interspace near the termination of the cartilages on the left side of the sternum, or in the epigastrium, along the free border of the ribs on the left side.

When dilatation of the right ventricle is extensive, with slight hypertrophy, the area of pulsation is more extended, and the character of the impulse is more diffused.

Dilatation of the right auricle may cause pulsation to be seen in the fifth right interspace along the sternum. Pulsation of the conus arteriosus and hypertrophy and dilatation of the right ventricle may produce pulsations in the second and third interspaces on the left side.

Cardiac weakness from any cause, as myocarditis, fatty heart, degenerations, etc., causes the apex beat to be less noticeable or entirely absent.

In congenital transpositions of the organs the apex beat is found to the right side of the fifth interspace, and has the characteristics of that noted in the normal heart.

Influence of Changes in the Mediastinum.—Aneurism of that portion of the aorta contained in the mediastinum, abscesses, new growths and enlarged glands displace the apex beat downward and to the left. Fibrinous mediastinitis with contraction may bind the heart down and cause retraction of the præcordial area during systole.

Influence of Abdominal Changes.—Pressure from below the diaphragm, due to ascites, meteorism, tumors or anything that increases the contents or the abdominal pressure forces up the diaphragm, causing the heart to be displaced upward and the apex beat to be seen in the fourth interspace to the left.

Displacement of the diaphragm downward, as occurs in ptosis of the abdominal organs, etc., carries the apex downward, and the impulse is seen in the epigastric region only.

IMPULSES DUE TO CHANGES IN BLOOD-VESSELS.

Aneurism of the thoracic aorta may produce visible pulsations in different portions of the thorax. When it involves the ascending portion, the pulsation is seen to the right of the sternum. When the transverse portion is involved, the impulse may be conveyed to the sternum itself and upward, and be seen also in the episternal notch. Aneurism of the descending aorta, when developing forward, shows the impulse to the left of the sternum.

Arterial Pulsations.—Normally, the only pulsation that is seen in the arteries is a slight movement in the carotids just above the clavicles.

Conditions Causing Visible Pulsations in the Arteries. *(1) Cardiac Causes.*—When cardiac action is sudden and forcible, as occurs in exercise and under mental excitement, the blood is forced into the aorta and larger blood-vessels with sufficient force to cause a marked visible pulsation.

(2) Vascular Causes.—When the blood is forced from the heart into the blood-vessels that are only partially filled, instead of the normal pulse wave being transmitted through the blood-vessels, there is a direct propulsion of the blood wave through the aorta into the blood-vessels themselves. This condition of the blood-vessels occurs *(a)* whenever there is a rapid emptying of the larger blood-vessels into the capillaries and veins, as occurs in vaso-motor loss of control, in Grave's disease, chlorosis and anæmia; *(b)* when the aorta, during diastole, empties itself into the enlarged ventricle (aortic regurgitation), and with the following systole there is a sudden forcing of a large amount of blood into the partially empty and collapsed aorta and its branches. In aortic regurgitation the visible systolic pulsation may be seen over the entire body. Arterial pulsation is also seen when the blood-vessels have been converted from elastic, distensible tubes into rigid ones. Normally, with each systole there is dilatation of the aorta, and during diastole the elasticity of the large vessels forces the blood onward. When the aorta is converted into a rigid tube, with each systole the heart impulse is conveyed to the entire arterial tract, producing visible movement in the blood-vessels.

Capillary Pulse.—Occasionally in aortic regurgitation systolic, rhythmical pulsations are seen in the capillaries; with loss of cardiac power these pulsations disappear. Capillary pulse may be observed by friction of the surface, producing dilatation of the superficial blood-vessels by placing a slide on the mucous membrane of the lips and with slight pressure causing the capillaries alone to be seen.

Venous Pulsation.—Slight pulsations may be seen in the neck during health, but are most marked in the subjects of chlorosis and anæmia. In order to see them, the patient should be recumbent. the head thrown slightly backward and the light should fall across the root of the neck obliquely. The physician should view the neck at a slightly higher level.

The *normal* venous pulsations are usually a gradual, general dilation of the blood-vessel. "The wave is longer, broader and less sudden in its oscillation than that seen in the artery. It is more influenced by posture, being greatest when the patient is lying flat. It is *pre-systolic,* occurring before that of the carotid; the collapse of the vein corresponds to the apex beat."

In chlorosis and anæmia this presystolic venous pulsation may be markedly increased. The *abnormal* pulsations seen in the veins are systolic in time. They are most marked in the veins of the right side, and are best looked for in the external jugulars just above the clavicle, outside of the border of the sterno-cleido-mastoid muscle, as at this point the vein is unaccompanied by the artery.

It is necessary to determine whether or not the systolic pulsation in the vein is due to the impulse conveyed to the vein from a distance. (1) If slight, slow pressure over the vein at the lowest point stops the pulsation, it shows that it is a true venous pulsation. Such a light pressure would not influence pulsations transmitted from the artery. (2) By drawing the finger upward along the course of the vein, and keeping it at the upper level, the vein will fill from below and the pulsation will be systolic. Sudden emptying of the vein during diastole is said to be a sign of adherent pericardium, but this is a doubtful sign.

Pulsation in the peripheral veins may be due *(a)* to aortic regurgitation; *(b)* certain stages of fever, and is made more visible by putting a thin layer of sealing wax over the vein.

Cardiac Dyspnœa.—Cardiac dyspnœa differs from pulmonary dyspnœa in the following respects: (1) While the patient is at rest the respiratory movements are normal, but the slightest exertion induces an intense feeling of oppression and dyspnœa. (2) Examination of the lung shows no interference with the entrance and exit of air; respiratory movements are increased in force, frequency and extent without the other evidences of interference in the respiratory tract. (3) The dyspnœa is out of all proportion to the lesion that may have been induced secondarily in the lung by the cardiac disease.

TYPES OF CARDIAC DYSPNŒA.—(1) Dyspnœa on exertion, (2) paroxysmal dyspnœa, occurring when at rest, especially when the patient wakes out of sleep, due to sudden raising of the blood pressure; (3) chronic dyspnœa or orthopnœa, (4) rhythmical dyspnœa (Cheyne-Stokes respiration).

Dropsy.—Œdema due to cardiac disease appears first over the dorsum of the feet and at ankles and skin over the lower portion of the tibia. As interference with the circulation becomes more marked, the dropsy ascends from below upward, involving the thighs, abdomen and finally becomes general (anasarca), when the serous cavities become involved.

Œdema due to renal causes appears first in the face, and soon becomes general. In anæmia the dropsy first develops in the more dependent parts of the body, and in this respect resembles that of cardiac disease.

CHAPTER IX.

PALPATION.

PALPATION not only corroborates the data obtained by inspection, but also corrects it, and gives additional and more exact information concerning the changes in the præcordia and their causes. It enlarges the knowledge of cardiac movements by detecting pulsations too feeble to be noticed by the eye, and shows not only the position of the apex beat, but also its extent, force, character and rhythm.

While inspection gives us the point at which some portion of the heart strikes the chest wall, by palpation we are able to fix the position of the anatomical apex, and it is thus possible to determine the true size of the heart.

It is especially valuable to determine whether pulsations are systolic, diastolic, irregular or intermittent, and to detect other vibratory phenomena, as fremitus and thrills.

In order to obtain the most satisfactory results from palpation, it is necessary that the patient assume an easy position, whether it be standing, sitting or reclining. When feasible, the patient should be examined in more than one position, as posture has a marked influence on the position and force of the cardiac impulse. The observer should place his hand lightly over the lower portion of the præcordia and note the general character of the sensation conveyed to it. Then, by the tips of the fingers, the exact location of the maximum apex beat may be determined. Frequently, when the pulsations are indistinct or diffuse, it is difficult to locate exactly the point of maximum intensity of the apex beat. In such cases it is best to apply the tips of the fingers to the thorax beyond the point of visible pulsation, and, moving them along the interspaces, locate the point at which the elevating impulse is felt most distinctly.

Palpation enlarges the knowledge obtained by inspection of increase in size of the præcordia by determining whether or not the enlargement is due to œdema, inflammatory swelling, or new growths of the soft parts. In regard to the bony thorax, it determines the degree to which it is influenced by each cardiac beat.

The cardiac movement is felt by the hand placed over the præcordia in the region of the apex, in an adult with fairly thin chest walls, as a diffused throb, which at the fifth intercostal space ends with a distinct elevating impulse. The pulsation felt is synchronous with the visible apex beat and the carotid pulsation, and slightly precedes the radial pulse. In thin persons with flexible chest walls, a distinct, sharp jerk or shock may be felt at the base of the heart when the flat of the hand is applied. With the finger tips it can be localized in the third or fourth interspaces, close to the sternum. It is diastolic in time, due to the closure of the aortic and pulmonary valves (valve shock). In its most perfect manifestation, cardiac impulse is felt as a distinct movement or peristaltic wave passing from apex to base.

The apex beat varies normally in its point of maximum intensity, force and extent, as has already been described under "Inspection."

Conditions Modifying the Apex Beat.—The influence of posture on the position of the heart is much more noticeable by palpation than by inspection, and must always be taken into account. When the patient is standing, bending forward causes the cardiac impulse to become more distinct and sustained. In many cases the apex beat can only be located with the patient in this position. When in the semi-recumbent position or lying flat upon the back, the heart recedes from the chest wall, and the impulse becomes correspondingly weak and indistinct. When lying on the left side, the heart is displaced toward the same side, and is felt about midway between its normal position and the anterior axillary line; and the apex impulse is not so marked, being covered to a greater extent by lung tissue. When lying on the right side, the heart is carried toward the right, and the apex beat is felt nearer to the sternum, or it may be under the sternum, and the impulse will be faint or entirely absent. When the heart is carried under the sternum, the beat may be sometimes felt as a diffused, weak epigastric impulse.

The position of the apex beat varies with age, as has already been explained. During the first two years of life it is visible in the fourth interspace, but palpation shows that the true apex is near or to the left of the nipple line. From the second until the sixth year it gradually assumes a position nearer to the median line and lower, until after the sixth year it is found in the fifth interspace. In childhood the force of the beat is relatively greater, on account of the flexibility of the ribs and the condition of the lungs. In old age the heart is displaced downward and toward the median line, and the apex beat is stronger on account of senile hypertrophy.

Influence of the Sort Parts.—Marked increase in the thickness of the thoracic wall has but a slight effect upon the palpable apex beat; by firm pressure we are able to render the tissues in such a condition as to readily convey the impulse.

Influence of the Bony Thorax.—In young subjects, with thin, elastic ribs, the cardiac impulse is sufficient to be felt as a lift or elevating sensation, not only in the intercostal spaces, but also over the ribs, the point of maximum intensity of the apex beat being much more distinct and less diffused than in the adult. Where the bony structures are rigid, the cardiac impulse influences the bony thorax but slightly, and the apex beat is only

Adhesions of the pleural surface and contractions draw the heart toward the affected side, with displacement of the apex beat. This will be fully explained later on.

Influence of Changes in the Lung. — Quiet breathing produces no change in the apex beat. Full, deep inspiration causes the inflated lung to cover the heart to a greater degree, thus lessening the extent and force of the apex beat, and may cause it to disappear entirely. Frequently it is sufficient to displace it downward, so that it is seen an interspace lower.

Permanent increase in size of the lungs, as in general emphysema, causes marked change in the relation of the heart to the bony thorax. As a result of the increase in size of the lungs and loss of tension, as was explained under "Emphysema," there is elevation of the ribs and depression of the diaphragm, which also depresses the heart, the apex beat being carried inward and downward. When increase in size of the lung is associated with obstruction of the circulation, the hypertrophy and dilatation of the right ventricle increase the pulsation in area and extent, and carry it still lower, giving epigastric pulsation.

Unilateral emphysema causes the heart to be displaced similarly to the manner seen in pleurisy. Compensatory emphysema in the right lung carries the apex beat to the left and higher. Emphysema of the left lung causes it to be lower, and rarely displaces it beyond the left sternal line.

Shrinkage of the lung as a result of pleurisy, or due to interstitial change in the pulmonary tissue (as in interstitial pneumonia and fibroid phthisis), draws the heart toward the diseased side. When the changes are limited to the lower border of the left lung, that portion of the heart which is normally covered by pulmonary tissue may be brought into direct contact with the chest wall; the visible apex beat will be increased in area and carried horizontally to the left, and a greater portion of the heart, including the true apex, strikes the chest wall. When the change is more extensive, the heart may be drawn upward and outward, the apex beat being seen in the mid-axillary line. When the right lung is affected, the heart is drawn to the right side and upward. Involvement of the upper portion of the lung may cause visible pulsations of the pulmonary artery in the second left interspace, or it may be so extensive that the phases of cardiac movement, systole, diastole and the pre-systolic contraction of the auricle may be seen over the entire præcordial space, giving the impression of a peristaltic wave.

Influence of Changes in the Pericardium.—Effusion into the pericardium causes change in the site and area of the apex beat, according to the character and amount of exudation. In small effusions the apex beat may be seen over a wider area, especially toward the epigastrium. When the pericardium is filled to a greater degree, the visible apex beat is carried upward into the fourth interspace or higher, due to a portion of the heart higher up striking the chest wall, and not to an upward displacement of the true apex. When the pericardium is fully distended with fluid, the apex beat may be absent.

Adhesions of the pericardium to the pleura or mediastinum may cause retraction of the chest wall over the apex (Broadbent's sign). Retraction of the chest wall over the præcordial area is one of the diagnostic signs of adhesions of the pericardium when it produces marked visible recession of the costal cartilages, ribs and sternum, and is associated with signs of cardiac hypertrophy greater than the amount of cardiac valvular disease would produce, and where there is also evidence of a co-existing pleurisy or exo-cardial disease.

Retraction of the chest wall over the apex also occurs with adherent pericardium, secondary to pulmonary tuberculosis.

Influence of Changes in the Heart.—The site, extent and character of the apex beat corresponds, within certain limits, to the extent of the cardiac changes. Powerful action of the heart, due to physical exertion or mental excitement, as in Grave's disease, in poisoning and in fever, causes the apex beat to be more noticeable and broader by causing a larger area of the chest wall to be affected.

In a similar manner, simple hypertrophy of the left ventricle causes the apex beat to be carried to the left and very slightly downward. Increase in the size and force of the left ventricle causes a portion of the heart closer to the true apex to strike the chest wall, and the beat is strong, distinct and defined. When the left ventricle is both dilated and hypertrophied, the apex beat is carried downward and to the left, being seen in the eighth interspace or lower. The impulse is in proportion to the amount of hypertrophy; the apex beat will be correspondingly strong, a large area of the chest wall will be influenced and the impulse, though wide, will be well defined.

When dilatation is the most marked feature, the apex beat will be weak and diffused.

Right-sided cardiac hypertrophy causes the visible pulsation to appear between the ensiform cartilage and the normal position of the apex beat in the fifth interspace. The apex beat may be carried slightly to the left, but rarely goes beyond the left mammary line. When the hypertrophy of the right ventricle is extensive and associated with dilatation, pulsation may be seen in the third, fourth, fifth, sixth and even seventh interspace near the termination of the cartilages on the left side of the sternum, or in the epigastrium, along the free border of the ribs on the left side.

When dilatation of the right ventricle is extensive, with slight hypertrophy, the area of pulsation is more extended, and the character of the impulse is more diffused.

Dilatation of the right auricle may cause pulsation to be seen in the fifth right interspace along the sternum. Pulsation of the conus arteriosus and hypertrophy and dilatation of the right ventricle may produce pulsations in the second and third interspaces on the left side.

Cardiac weakness from any cause, as myocarditis, fatty heart, degenerations, etc., causes the apex beat to be less noticeable or entirely absent.

In congenital transpositions of the organs the apex beat is found to the right side of the fifth interspace, and has the characteristics of that noted in the normal heart.

Influence of Changes in the Mediastinum.—Aneurism of that portion of the aorta contained in the mediastinum, abscesses, new growths and enlarged glands displace the apex beat downward and to the left. Fibrinous mediastinitis with contraction may bind the heart down and cause retraction of the præcordial area during systole.

Influence of Abdominal Changes.—Pressure from below the diaphragm, due to ascites, meteorism, tumors or anything that increases the contents or the abdominal pressure forces up the diaphragm, causing the heart to be displaced upward and the apex beat to be seen in the fourth interspace to the left.

Displacement of the diaphragm downward, as occurs in ptosis of the abdominal organs, etc., carries the apex downward, and the impulse is seen in the epigastric region only.

IMPULSES DUE TO CHANGES IN BLOOD-VESSELS.

Aneurism of the thoracic aorta may produce visible pulsations in different portions of the thorax. When it involves the ascending portion, the pulsation is seen to the right of the sternum. When the transverse portion is involved, the impulse may be conveyed to the sternum itself and upward, and be seen also in the episternal notch. Aneurism of the descending aorta, when hand, while the fore finger of the right hand is applied longitudinally over the artery. In this way the vessel is felt over a considerable length of its course by one finger of the observer, and varying degrees of pressure can be applied to it, while the skin can be pushed up and the size of the artery and the character of the arterial coats can be determined." (Sanson.)

The sensation that is felt when an artery is palpated is due to the momentary increase in arterial pressure that occurs when the left ventricle empties itself into the aorta. The pulse wave distends the vessel that has been partially empty during diastole, and whose lumen is still further narrowed by the examining finger. "The pulse then indicates simply the degree of duration of the increased pressure in the arterial system caused by the ventricular systole." (Broadbent.)

It is necessary to bear in mind that three factors are concerned in the production of the pulse: (1) Cardiac action, which determines *(a)* frequency (fast or slow), *(b)* force (strong or weak), *(c)* rhythm (regular, irregular or intermittent).

(2) The elasticity of the blood-vessel, upon which depends the degree of compressibility of the pulse (hard or firm, compressible or incompressible).

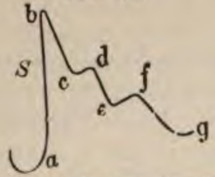
(3) Resistance in the arterioles and capillaries, which determines the readiness with which the larger blood-vessels are emptied during the period of diastole and regulates *(a)* the size of the pulse; whether the excursion of the artery compressed by the finger is wide or narrow, when the pulse is spoken of as large or small; *(b)* the duration of the time that the wave is perceptible to the touch, so that the pulse is long or short; *(c)* tension or the degree of distension of the blood-vessels present throughout the cardiac diastole, and especially near its end.

Size, duration and tension are usually associated in a definite manner. Low resistance in the artery and capillaries give the pulse of large size, short duration and low tension, while great interference causes the pulse to be small, long and of high tension.

These three factors are more or less correlated and react on each other, and each exerts its own peculiar influence on the pulse, both in health and disease.

The Normal Pulse.—The normal pulse for the healthy adult male beats 72 times per minute (frequency); the strength of each distending impulse is the same (force), and the beats follow each other at regular intervals (rhythm). The lumen of the artery is readily obliterated by the examining finger nearest the heart, and the pulse wave is stopped at the point of pressure, beyond which, distally, the artery cannot be detected as a separate structure. The pulse wave is felt as a dilating force of some volume (size), which does not raise the finger to the full height at once, but takes a definite time to reach its acme and declines in the same manner (duration); and between the pulsations the artery is felt under the finger, and can be compressed with greater or less ease according to its degree of distension (tension).

FIG. 29.



Sphygmographic tracing of normal pulse.
a, *b*, percussion up-stroke; *a*, *b*, *c*, percussion wave; *c*, *d*, *e*, tidal wave; *e*, *f*, *g*, dicrotic wave; *d*, *e*, *f*, aortic notch; *f*, *g*, diastolic period.

It is necessary to distinguish between the incompressibility of a blood-vessel due to condition of its walls and the amount of pressure necessary to overcome the distension of the vessel due to the blood pressure (tension). When the pulsations in the artery are obliterated with difficulty, if the incompressibility is due to increased thickness of the arterial coats, the artery will be felt beyond the point of compression as a more or less rigid cord; while if the incompressibility is due to high arterial tension, it will be impossible to detect the artery beyond the compressing finger.

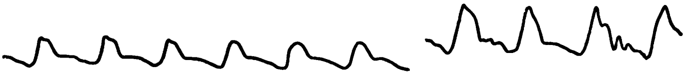
While the above are the average characteristics, each pulse may present individual peculiarities in the three factors spoken of. Both radial arteries should be examined, and when differences are detected it is necessary to determine whether they are due to anomaly in the size or distribution of the arteries, or are dependent upon pathological changes, as aneurysms, plugging of the blood-vessel, etc.

I. Conditions Modifying the Cardiac Elements of the Pulse. *(a)* FREQUENCY.—While the average pulse rate is 72 per minute, there are certain normal variations. Usually the pulse of females is slightly quicker—80 per minute—and it has been claimed that in tall people the pulse is slower. At birth the pulse is 120 to 140, and gradually diminishes in frequency until at six years of age it is 100, and at puberty 80 or less. In old age the pulse becomes slower, but in extreme age its frequency is increased. There is also a diurnal variation in the pulse rate, corresponding to the body temperature, it being more frequent in the afternoon and evening and after eating, slower during sleep and during the early morning hours. While a pulse of 60 may be normal for one adult and 100 for another, usually a pulse of over 90 per minute is morbid.

Increase in Frequency.—The rate of the pulse may be increased by the following: *(a)* Muscular exertion. *(b)* Mental and emotional excitement; the degree of increase will vary with the individual, according to the irritability of the nervous system (temperament). *(c)* Reflex irritation from other organs, especially the abdominal and pelvic viscera. *(d)* Diseases of the nervous system, especially those involving the nervous mechanism of the heart through the pneumogastric, sympathetic nerves or the cardiac ganglia. *(e)* Fever. As a rule, for each degree of temperature above the

Sphygmographic Tracings of the Pulse.

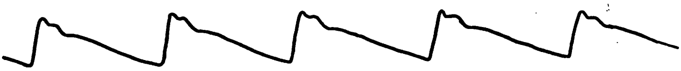
FIG. 30.



Right radial artery. **Left radial artery.**

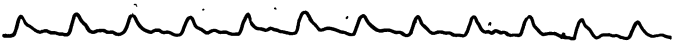
Aneurysm of arch of aorta.

FIG. 31.



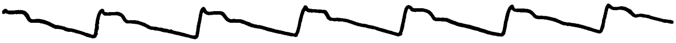
Slow pulse (60) (acromeglia).

FIG. 32.



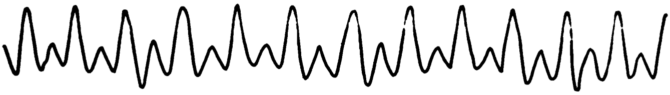
Small, thready, rapid pulse (98) (chill of malaria fever).

FIG. 33.



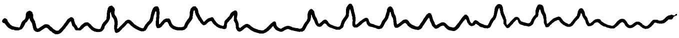
High-tension pulse (chronic nephritis).

FIG. 34.



Low-tension dicrotic pulse (pneumonia, eight hours before death).

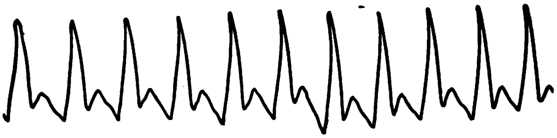
FIG. 35.



Low-tension pulse, feeble cardiac action (typhoid fever, third week).

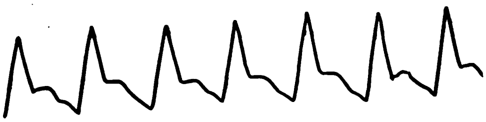
normal there is an increase of eight or ten beats. Certain diseases do not conform to this rule; thus in typhoid fever the pulse is generally slow in proportion to the temperature, and is of diagnostic value in differentiating this disease from tuberculosis and septic conditions. When, in typhoid fever, the pulse rate corresponds to the temperature range, it indicates a grave condition. In scarlet fever, with the very beginning of the throat symptoms, the pulse ranges from 120 to 160, and this is of great aid in differentiating scarlatinal sore throat from that due to other causes, as diphtheria, simple follicular tonsillitis, etc. In fevers

FIG. 36.



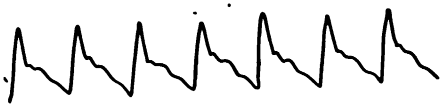
Rapid (100) low-tension, dicrotic, on admission.

FIG. 37.



Twelve hours later (80), free stimulation, slower (88).

FIG. 38.



Six hours after crisis (86). Four days later, no stimulation.

Pneumonia, showing variation in pulse.

due to septic infection, the pulse is accelerated out of proportion to the range of temperature, and in the puerperium a rapid pulse, with slight elevation of temperature, is a suspicious sign. *(f)* Anæmia and debility. *(g)* Respiratory diseases. In nearly all of the acute inflammatory diseases of the respiratory tract the pulse rate is increased. Normally, the ratio between pulse and respiration is 4 : 1. This ratio is disturbed in respiratory diseases, such as pneumonia, when the ratio becomes 3 : 1, or even 2 : 1; or equal. In febrile conditions due to pulmonary disease, this sign is found of value when the usual physical signs are absent. *(h)* Cardiac conditions. The pulse may be accelerated in all valvular diseases, although as long as compensation is perfect the increase may be very slight. When there is muscular insufficiency, the rate is markedly increased, especially upon exertion.

Acceleration of the pulse is a prominent symptom of neuroses of the heart. In Grave's disease the pulse ranges from 100 to 120; in paroxysmal tachycardia it may reach 160 or 200, but does not remain permanently at this point, although it may be constantly above the normal. In irritable heart (paroxysmal hurry) it is also increased.

Rheumatoid arthritis, before marked deformity of the joints, frequently has a pulse, as an early symptom, of 110 or 120.

The rate of the pulse bears a definite relation to the amount of tension in the blood-vessels, and should always be considered in this relation.

Diminution in Frequency.—As mentioned before, the pulse may be habitually slow as an individual peculiarity. Usually a pulse under 60 is considered morbid, while a pulse of 40 is usually of grave significance.

The pulse may be slowed by *(a)* reflex nervous irritation. *(b)* Nervous diseases, especially those which cause increase in the intercranial pressure, as tumors, hemorrhage and meningeal effusion. It is also slowed in certain cases of mania, melancholia and general paralysis of the insane, and in injury to the cervical portion of the spinal cord. A slow pulse in epilepsy is always a grave symptom. In myxœdema the pulse is constantly slow. *(c)* Convalescence from fevers. *(d)* Toxæmias, especially bile, urea and certain acute diseases. *(e)* Increase in arterial tension. *(f)* Cardiac disease. A slow pulse is a grave symptom when it occurs in cardiac disease, and was formerly supposed to be pathognomonic of fatty degeneration of the heart, but is not usually present except when the degenerative change is due to atheroma of the coronary arteries.

The pulse is not always a true guide to the frequency of the heart, as a form of slow pulse occurs in which only every other beat of the heart is felt at the wrist. The examination of the heart shows that the beat which is not felt as a pulse in the artery is feeble and incomplete. This type of slow pulse is most frequently present in mitral stenosis. This form of infrequency often alternates with a form of irregularity called "pulsus bigeminus," as will be explained later.

(b) FORCE.—The force of the pulse wave is dependent on the energy with which the blood is forced by the ventricle into the aorta. It bears a certain relation to the force of the apex beat, so far as it (the apex beat) depends upon the muscular strength of the left ventricle. This rule holds good as long as the ventricle propels into the aorta the normal amount of blood in the normal time, as the fullness of the arterial system during the entire cardiac cycle is an important element in the strength of the pulse.

Increase in Force.—The force of the pulse wave is increased *(a)* when the cardiac action is more vigorous than normal, as occurs in exercise, in mental and emotional excitement, and in cardiac neuroses, as Grave's disease. *(b)* When the cardiac muscular power is increased above the normal, due to change in the muscle, as occurs in simple hypertrophy of the left ventricle. When, in addition to hypertrophy of the muscle wall, the cavity of the left ventricle is enlarged (dilatation), not only is the force of the blood wave augmented, but it has greater volume.

Diminution of Force.—The pulse wave may be decreased in strength *(a)* when the contraction of the cardiac muscle is less forcible on account of general muscular weakness, as in debility, or due to lack of nerve tension, as in neurasthenia, etc. *(b)* When there is true muscular weakness, as occurs in cardiac degeneration, which may be primary or secondary to valvular disease of the heart. *(c)* When the amount of blood that is forced into the aorta is decreased through imperfect emptying of the heart from cardiac weakness, from imperfect filling of the left ventricle, and general reduction in the amount of blood, as in hemorrhage.

Irregularity in Force.—In health the individual beats have the same volume. Under certain conditions there is inequality of force, some of the beats being fuller than others. This form of irregularity is present most frequently in mitral regurgitation, when dilatation and weakness of the left ventricle occur; it may be made more pronounced by raising the arms above the head, increasing the action of gravity. Its presence should always direct attention to the condition of the cardiac muscle. When irregularity in force is due to muscular insufficiency, slight exertion is often sufficient to induce also irregularity in rhythm.

(c) RHYTHM.—The normal rhythm of the heart may be disturbed in two ways: *(a)* A beat is dropped at more or less regular intervals, giving the intermittent type of pulse. *(b)* The beat occurs at irregular intervals, giving the true irregular or arrhythmical pulse.

Intermittent Pulse.—Intermittent pulse has the characteristic that, with the exception of the missing beat, the pulse is regular. The intermission may occur according to rule; that is, after a definite number of beats, as when the fifth, seventh, eleventh or twenty-first beat is lost; or it may follow no rule. Examination of the heart may show a corresponding omission of the cardiac systole (pulsus deficiens), or that there is a cardiac systole, but too weak to be felt at the radial artery (pulse intermittens).

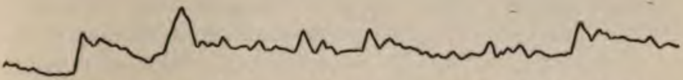
When the intermittent pulse is habitual, the intermission is apt to be according to rule. This form is of little clinical significance, being often a constitutional peculiarity; or it may be caused by the use of tea, coffee, tobacco, etc. It must be borne in mind that the intermission usually disappears during pyrexia from any cause, and returns with the normal temperature. When it returns during the height of the temperature, it is an unfavorable sign.

In degeneration of the myocardium and in valvular diseases the pulse may be intermittent while the patient is at rest, but on exertion the intermission is replaced by true irregularity, with shortness of breath and other signs of cardiac insufficiency. When the intermission is due to some disturbance of innervation, on exertion such change does not occur, or the rhythm may become perfectly regular. Broadbent claims that an intermittent pulse is not a contraindication to the administration of anæsthetics (chloroform), but that the condition of the pulse should be watched during the early stage of narcosis. Generally the intermission disappears, but if from the first the pulse becomes *weak and irregular,* the administration of the anæsthetic should cease. The same rule applies to ether and nitrous oxide.

Irregular Pulse.—Irregular pulse has the characteristic that the beats follow each other at irregular intervals, and are also irregular in force. The tendency to irregularity varies at different periods of life. During infancy the pulse is easily disturbed in all its cardiac elements, especially its rhythm. The same tendency to arrhythmia is noted in extreme old age. Irregularity of the pulse may be a normal condition in individuals when it is constant.

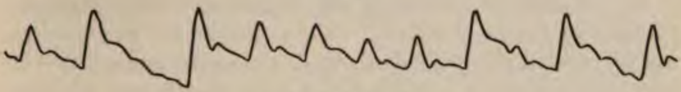
The causes of irregularity may be: *(a)* Reflex irritation from abdominal or pelvic viscera, especially when they are associated with flatulency and displacement

FIG. 39.



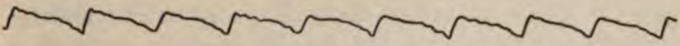
Pulse irregular in rhythm and force (myocarditis with dilatation).

FIG. 40.



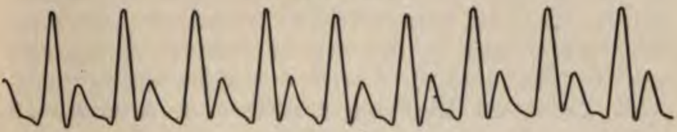
Mitral regurgitation.

FIG. 41.



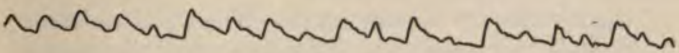
Aortic obstruction.

FIG. 42.



Aortic regurgitation.

FIG. 43.



Mitral stenosis.

upward of the diaphragm. *(b)* Disturbances of the nervous system by shock, grief, continued worry, neurasthenia, anæmia, debility, etc. *(c)* Diseases of the brain and meninges. *(d)* Toxæmia, due to alcohol, tea, coffee, tobacco, digitalis, belladonna, etc., or that of the infectious diseases, especially the early stages. The irregularity that occurs later in acute disease may be due to the toxæmia or to degenerative changes in the cardiac muscle. *(e)* Cardiac diseases. In valvular disease marked irregularity does not occur during stage of compensation, but is one of the early signs of muscular insufficiency. In mitral stenosis it is a common, but not grave, symptom. In all forms of myocarditis it is an important prognostic sign.

II. Conditions Modifying the Elasticity of the Arteries.—The sensation conveyed to the finger by the normal artery has been described, and attention called to the necessity of discriminating between the degree of compressibility due to changes in the coat of the artery and that due to increased blood pressure. When the coats of the arteries are thickened and rendered inelastic by increase of fibrous tissue, the pulse wave will be obliterated with difficulty, and beyond the point of compression distally the artery will be easily detected as a more or less rigid cord. As the tube becomes more inelastic, it becomes longer and tortuous, and each pulse wave is felt, not as a beat, but as a worm-like movement. Calcareous deposits may be detected as isolated plates, and "may feel like a string of beads under the finger."

III. Conditions Modifying Resistance in Arteries and Capillaries.—The amount of arterial and capillary resistance determines the size, intensity and duration of the normal pulse, and any change in the degree of resistance influences all of these elements.

Diminution of Resistance.—When the resistance is reduced below normal, there is a relatively greater increase in the distension of the artery with each ventricular systole. The pulse wave passes more rapidly under the finger, and between the beats the artery is but slightly distended with blood, and is almost obliterated by light pressure of the examining finger; and the pulse felt at the wrist has the following characteristics: The pulse wave dilates suddenly the flattened artery, giving the sensation of a large-sized pulse; it does not gradually reach its acme, but seems to reach the finger full size. The beat is sharp, passes quickly under the finger (short duration) and the artery remains distended but for a very short time (tension rapidly falls). This type of pulse is spoken of as "large, short, low-tension pulse."

In the normal pulse the examining finger can scarcely detect dicrotism, which is caused by the elastic recoil of the artery on the closure of the aortic valves, although it is very perceptible in the sphygmograph tracing. (Fig. 29.)

When the resistance in the arteries is very low, this wave becomes quite apparent to the finger, and the pulse is spoken of as dicrotic. In low tension the force and frequency of the heart determines the type of pulse. When the heart is beating rapidly and forcibly, the arteries are kept well filled, the pulse wave is felt as a large, sharp, well-sustained beat, and the pulse is called "full and bounding," and is easily obliterated. Dicrotism, while present, is not noticed, on account of the size of the primary pulse wave. As the force of the heart diminishes, the pulse becomes less full, and is sharper and shorter and more easily obliterated by light pressure on the artery, and the dicrotic wave becomes more pronounced. This type of pulse is spoken of as the "gaseous pulse." When the heart power becomes insufficient to empty the left ventricle, the pulse is weak and characterless—"small, running pulse" (page 245).

Causes of Low Tension.—*(a)* Low tension may be a constitutional peculiarity in some individuals and families, when it may be associated with increased frequency in some cases and obesity and weak cardiac action in others (diminution in capillary resistance). *(b)* Temporary low tension is caused by warmth, as weather, baths, hot drink and food. *(c)* It may be dependent upon nervous causes, as mental or nervous exhaustion from whatever cause. *(d)* Debility due to malnutrition or secondary to chronic disease. *(e)* Pyrexia generally causes a full, bounding pulse, usually in proportion to its degree. Later the pulse may become weak, quick and dicrotic, on account of the effect of the high temperature on the cardiac muscle or the influence of the toxines on the heart itself, or on the nervous system. *(f)* Cardiac diseases. As the pressure in the arterial system depends upon the amount of blood that is forced into it, as well as the resistance to the passage of the blood through the arterioles and capillaries, the tension will be lowered whenever the heart is unable to empty itself, due to valvular disease or to muscular weakness. In aortic insufficiency the collapsing or water-hammer pulse is due to the sudden fall in the blood pressure caused by the free regurgitation of blood into the ventricle. The peculiarities of this pulse will be described under aortic regurgitation.

Increase of Resistance.—In normal conditions there is a certain correlation between the force and frequency of the heart and the outflow from the arterioles, and a normal mean blood pressure is maintained. The normal blood pressure varies at different periods of the cardiac cycle. With cardiac systole, the pressure is raised to the maximum, and the artery is felt as a tense, distended tube. During diastole, as the pressure gradually subsides, the tube becomes lax, and can no longer be felt. When resistance to the outflow of blood is increased, the tension is increased, the pressure in the blood-vessels does not subside; and the artery remains filled, so that it is detected not only with the beat, but in the interval. The sensation conveyed to the finger by the pulse wave also is changed. The examining finger not causing so marked a flattening of the artery, there is less motion felt, and the pulse wave slowly reaches its height to slowly decline. As the size of the artery is not much altered between the pulse waves by light pressure, the pulse is small, and the wave gradually distends the vessel and is felt for a relatively long period, and the artery remaining perceptible in the interval.

As in low tension, so in increased resistance the type of the pulse will vary with the force and frequency of the heart and the condition of the large blood-vessels. When the muscular power of the heart is sufficient to overcome the resistance in the blood-vessels, and the muscular coats of the aorta oppose the increased cardiac action, the tension in the arteries will be sustained almost uniformly during the interval, and the pulse will be correspondingly small, long and of high tension.

When the heart fails, the pulse becomes irregular and arterial pressure is lowered. When, on the other hand, the aorta and large vessels yield to the blood pressure and become dilated, the pulse becomes larger and the duration is shorter, although the arteries remain overfilled during diastole.

Causes of High Tension.—*(a)* Temporary high-tension pulse may be caused by increase in the amount of blood, as occurs for a short time after meals or after taking large quantities of water, and is persistent when the elimination of the fluid by the skin and kidneys is interfered with, producing permanent overdistension of the blood-vessels. *(b)* It may be also temporarily increased by frequent and powerful overaction of the heart, as occurs in mental or emotional excitement and in the early stage of physical exertion. Or *(c)* it may be due to temporary increased resistance in the blood-vessels, as occurs from external cold, congestive chill, or hysteria.

Permanent high tension may be caused by all those factors which cause increased resistance in the capillaries, *(a)* as drugs, ergot and the digitalis group; *(b)* toxæmia, as occurs in gout, rheumatism, lead poisoning and in renal disease; *(c)* changes in the walls of the capillaries which interfere with permeability, as atheroma and arterio-capillary fibrosis. These changes may be primary or secondary.

CHAPTER X.

PERCUSSION.

By percussion of the cardiac region it is possible *(a)* to determine the size of the heart in certain dimensions only; *(b)* its relation to other thoracic and abdominal organs; *(c)* changes in the outline of certain portions.

Percussion of the præcordia gives two areas: (1) The area in which the clear pulmonary resonance becomes more and more impaired by the presence of the airless structure of the heart; that is, the area of relative cardiac dullness. (2) The area in which no pulmonary resonance can be detected, the area of absolute cardiac dullness or cardiac flatness. The percussion outline of these two areas is most difficult to determine. Many claim that they have no value, or, at least, only a limited value in the diagnosis of cardiac diseases.

The amount of information obtained by percussion over the præcordia depends upon the technique and the acuteness of the examiner, and whether or not the examination is conducted methodically and for a definite purpose.

Methods of Examination.—In percussing for relative dullness, it is most important that the line should be determined at which the normal pulmonary resonance is impaired by the underlying heart.

Percussion is begun over the thorax at some distance from the anatomical borders of the heart. The finger is placed across the ribs parallel with the sternum, and forcible percussion is made, the finger being moved inward toward the cardiac area till slight change in the percussion note is detected. This point is marked on the thorax, and the same area is again percussed with blows varying in strength, with the finger both in the vertical position and also parallel to the ribs lying in the interspaces, until the positive point of cardiac dullness is determined. This method is carried on at different levels, until the outermost area of cardiac dullness is outlined.

After this has been determined, the examiner then proceeds to outline the point at which the pulmonary resonance ceases and the heart, in contact with the chest wall, causes a perfectly flat note. It is necessary that the percussion blows vary in force as we proceed from the outer line of cardiac dullness, where the layer of lung is relatively thick, toward that of cardiac flatness.

The Area of Cardiac Dullness.—In the normal adult the outer area at which cardiac dullness is detected is as follows: On the right side no change is noted above the third rib, except occasionally a slight loss of resonance due to the underlying aorta. Although the right auricle projects beyond the sternum opposite the third costal articulation, it is impossible to accurately map out the border which extends beyond the sternum, as the thickness of the pulmonary tissue is so great that percussion, sufficiently strong to penetrate to the underlying heart, will set up lateral vibrations also, which, being conveyed to the sternum, give a resonant note. The most that can be detected on the right side by skillful percussion and a trained ear is a slight change in resonance, extending from the fourth rib to the area of liver dullness.

On the left side, that portion of the heart that lies above the third rib cannot be detected by percussion, on account of the thickness of the pulmonary tissue and its close proximity to the sternum. At the lower level of the third rib, close to the sternum, the deep cardiac dullness is detected. The line of dullness extends in a curved line across the third intercostal space to the middle of the fourth rib, 3½ to 4 inches from the mid-sternum and just inside the nipple line. From this point it runs almost vertically downward to the sixth rib, just inside the nipple line. When the heart and structures contained within the mediastinum are normal, they do not influence to any appreciable degree the resonance of the sternum. (Fig. 22.)

The Area of Cardiac Flatness.—That portion of the heart that lies in direct contact with the chest walls gives a perfectly flat sound on percussion, and corresponds in outline to the anterior margin of the left lung. The resonance of the sternum prevents that portion of the heart that lies between the anterior edge of the right lung and the left edge of the sternum being detected by percussion. The thin lingula pulmonalis that covers the apex of the lung does not modify the flat percussion note. (Fig. 21.)

The normal area of cardiac flatness is somewhat quadrangular in shape. The right border extends along the left edge of the sternum, from the upper edge of the fourth rib and along the border of the sixth rib, where it merges into the hepatic flatness.

The upper borders run diagonally across the fourth rib for about two inches, and then vertically downward to the sixth rib. Accurate mapping out of the borders of cardiac dullness and flatness being very difficult, some authors have recommended that the area of cardiac flatness be estimated in two directions only.

(1) The vertical line. Percussion is made along the para-sternal line, one inch to the left of the sternum, and the following varieties of resonance are noted on percussion over the normal heart: *(a)* From the clavicle to the upper border of the third rib full pulmonary resonance is detected. *(b)* From the third rib there is a gradual diminution of resonance and a heightening of pitch (cardiac dullness), until at the upper border of the fourth rib there is absolute loss of resonance (cardiac flatness). *(c)* From the upper border

of the fourth rib the sound is flat, and merges, below the sixth rib, into that of the liver. Over this normal flat area very forcible percussion may detect a deep-seated, tympanitic resonance when distension of the stomach or intestines is present.

(2) Transverse line. Percussion is made along a line which corresponds to the upper edge of the fourth rib on the left side. The sound is flat for 2 to 2½ inches from the edge of the sternum, when it merges gradually into dullness, which terminates just inside the nipple line.

Numerous modifications of the ordinary method of percussion by the finger have been devised in order to increase the ability to outline the heart. The most important are: *(a)* Auscultatory percussion (page 94) and *(b)* plessimetric percussion or Sanson's method. Instead of the finger, a pleximeter of vulcanite is used. This pleximeter consists of two thin, flat, oblong plates, one 1 x ½ inch, the other ¾ x ⅜ inch, joined in the middle by a central square column 1½ inches high. It is closely held to the chest wall by the index and middle fingers of the left hand, placed on either side of the vertical column. "The observer commences by applying the pleximeter with its long diameter parallel to the sternum, about midway between the axilla and the right sternal border, percussion being made upon the summit of the column by one or two fingers of the right hand, the strokes from the wrist being sharp and decisive, though not necessarily forcible. This procedure in the normal conditions elicits the normal thoracic vibrations. The pleximeter is then approached nearer and nearer—always in parallel lines—to the sternum, until the line is reached where the vibrations are sensibly modified. By slightly inclining the pleximeter so that the vibrations come from its left edge, there is practically a line of surface without noticeable breadth. The point at which the resonance is modified is marked. By repeat ing the process just described at higher and lower lev and uniting the marks, the observer delineates the area on the right side of the outer border of the heart dullness. In like manner, by percussing from above downward, with the long diameter horizontal instead of vertical, the upper limit of the liver, as indicated by an area of modified vibrations, is arrived at."

In percussing on the left side of the chest, the observer begins at the left of the second rib, 2 or 3 inches to the left of the sternal border, and approaches to the right until the line of modified vibration is arrived at, which will be nearly parallel with that obtained on the right side.

Percussion is made at different levels, as on the right side, until the heart is outlined.

By this method not only is the outer border of the heart noted by the ear, but also detected by difference in vibrations, as felt by the finger resting on the lower plate. A certain amount of experience is needed in order to obtain the best results by this method.

Conditions Modifying the Normal Areas of Cardiac Flatness and Dullness.—Both areas may be proportionately increased or diminished, or the dimensions of one may be altered at the expense of the other.

NORMAL VARIATIONS.—The size and location of the percussion areas are influenced *(a)* by the age of the individual; *(b)* by the posture of the patient; *(c)* by the condition of the lungs according to forcible inspiration and expiration; *(d)* by the position of the heart due to elevation or depression of the diaphragm.

In children the heart is relatively larger than in adults, and lies higher in the thorax, while the greater elasticity of the lungs and flexibility of the bony thorax causes a much larger portion of the heart to be uncovered by lung tissue and in closer contact with the chest wall. In children, during full inspiration, the area of cardiac dullness is relatively increased, while that of flatness is diminished; and the reverse condition obtains during shallow breathing and full expiration.

The elasticity of the sternum and costal cartilages permits dullness to be detected at the right border of the sternum. The upper border of cardiac dullness reaches the middle of the second intercostal space in the parasternal line, and the outer left border to the nipple line at the third interspace. At the lower border of the fourth rib it extends a quarter to a half inch beyond the nipple line. The shape of the area of cardiac dullness is somewhat semicircular, with the convexity upward. The area of absolute flatness in children is also increased in extent and raised by the width of an interspace. The right border is close to the left sternal margin; the upper line is in the third interspace, and extends almost straight across to the upper border of the third rib, close to the mammary line. The left border is usually just within this line, and extends to the lower edge of the fifth rib.

It is necessary to bear in mind the variations in the cardiac percussion area that occur in children, as they have an important bearing on the diagnosis of effusions into the pericardium.

In old age the areas are smaller and lower in position. In the left para-sternal line, the upper limit of dullness is about the lower border of the fourth rib, and of flatness about the middle of the fifth rib. The left border is also nearer the median line.

The position of the patient alters in a slight degree the percussion areas. They are the same whether the patient is lying on the back or in the upright position. When lying on the right side, the area of flatness to the left of the sternum diminishes or disappears entirely. A smaller area of flatness may be detected just to the right edge of the sternum, in the third interspace. When lying on the left side, the area of flatness is increased, the left border being carried nearly to the mammary line.

Influence of the Soft Parts.—The effect of the soft parts on the resonance from the thorax has already been considered. When the thorax is thickly covered, it is impossible to accurately outline the area of cardiac flatness, that obtained by light percussion being generally carried too far to the left.

Influence of the Bony Thorax.—The elasticity of the bony thorax also exerts an influence on the percussion areas. As before explained, the highly elastic thorax of the child will give a much more accurate outline of the heart than the rigid and more resonant thorax of the adult.

Influence of the Lung and Pleura. — As the area of cardiac flatness corresponds to that portion of the heart that is uncovered by lung tissues and the area of relative dullness to the part covered by a thin layer, both areas will be increased or diminished by changes occurring in the lung or pleura. The normal variations in size occur, as has already been mentioned, during the ordinary acts of respiration. Enlargement of the lung, as occurs in general emphysema, causes a smaller part of the heart to be in contact with the chest wall, increases the thickness of the layer of lung covering the heart, and displaces the heart downward and toward the median line. These changes cause the area of cardiac flatness to be diminished or absent. As emphysema is usually attended with hypertrophy and dilatation of the right side of the heart, the areas of percussion dullness and flatness do not correspond to the size of the heart, so that a normal area of cardiac flatness, occurring with other physical signs of emphysema, is always indicative of a marked right-sided cardiac hypertrophy and dilatation. In compensatory emphysema of the left lung, both cardiac dullness and flatness to the left of the sternum may be diminished or absent. When the heart is displaced to the right, an area of varying dullness may be detected to the right of the sternum. In compensatory emphysema of the right side, pulmonary resonance extends to the left of the sternum, and the right border of cardiac flatness is *moved* further from the left sternal margin.

The area of cardiac flatness may be enlarged in all directions by retraction or consolidation of the anterior border of the lung, or thickening of the pleura. Retraction of the anterior border of the left lung, by uncovering the heart, increases the area of flatness at the expense of that of dullness, the shape of the area of flatness corresponding to changes in the lung and secondary displacement of the heart.

When the anterior border of the right lung is retracted, cardiac dullness may be detected to the right of the sternum. When the heart is displaced to the right, the degree of dullness will depend upon the extent to which the heart is in close contact with the chest wall.

Increase in the area of flatness over the præcordia may be due to changes in the pleura or to consolidation of that portion of the lung overlying and surrounding the heart.

It is impossible by percussion alone to determine where cardiac flatness ceases and that due to pulmonary or pleural change begins.

Effusion into either pleural investment may cause some enlargement of the cardiac area of flatness.

Influence of Changes in the Pericardium.—Changes in the pericardium increases the area of flatness either by causing hypertrophy of the heart (adhesive pericarditis) or distension of the sac (effusion). Fluid in the pericardium, collecting in the most dependent portion of the sac, causes *(a)* increase in the transverse diameter of flatness and marked dullness to the right of the sternum, beginning in the fifth interspace on the right side. On the left side the flatness extends upward and toward the nipple line. As the fluid increases in amount the shape of the area of flatness over the præcordia becomes more triangular, with a broad base below and blunt apex above. The amount of fluid determines the size and shape of the triangle, and causes the area of deep-seated dullness to be replaced by flatness, surrounded by a zone of tympanitic pulmonary resonance.

The resonance over the sternum is also changed when the fluid in the pericardium exerts pressure on it. Pneumo-pericardium causes the cardiac flatness to be replaced by a low-pitched tympanitic resonance.

Influence of Cardiac Changes.—Increase in the size of the heart, whether due to hypertrophy or dilatation, or both, increases the area of cardiac dullness to a much greater degree than that of absolute flatness, which, as before shown, is generally increased by causes external to the heart.

The size and shape of the increase in cardiac dullness, or flatness due to cardiac hypertrophy and dilatation, vary according as the right or left side is affected. Hypertrophy of the left ventricle causes the left border of both dullness and flatness to be moved to the left and slightly downward. The upper border remains fairly constant, while the right border is unchanged. The area of dullness over the apex is pointed. When dilatation and hypertrophy are associated, the area of flatness is more quadrangular; the transverse diameter at the level of the fourth rib is increased by extension toward the left, and the outline over the apex is rounded. In extreme dilatation and hypertrophy of the left heart, as occurs in insufficiency of the aortic valves, the left border of flatness may reach the anterior axillary line, and dullness the mid-axillary line.

In hypertrophy or dilatation of the right side, when the ventricle alone is involved, the upper border of flatness at the level of the fourth rib shows no marked increase. The lower left border is carried slightly outward, and the right border remains at the left margin of the sternum.

When dilatation and hypertrophy affect both ventricle and auricle, the dullness may be detected beyond the right border of the sternum as high as the fourth costal cartilage. Dilatation and hypertrophy of the right heart may cause slight change in the sternal resonance, but not to the same degree as pericardial effusion, aneurism, or mediastinal tumors.

Percussion of the Aorta.—The great vessels normally give very slight and hardly perceptible dullness in the second interspaces. When any dullness is detected, it is most marked on the right side and is rounded in outline. Increase of this area is generally due to aneurismal dilatation of the aorta, although mediastinal new growths can cause it.

CHAPTER XI.

AUSCULTATION.

Auscultation of the heart furnishes information concerning the normal cardiac sounds, their relative *(a)* rhythm, *(b)* character or quality, *(c)* points at which they are most distinctly heard (maximum intensity), *(d)* areas over which they are audible (areas of diffusion), *(e)* variations and modifications, both normal and abnormal; *(f)* abnormal or adventitious sounds (murmurs, frictions), etc., and the degree to which they replace the normal cardiac sounds.

Auscultation should always follow inspection, palpation and percussion, as the data obtained by these methods are differentiated and enlarged by those detected by auscultation, while at the same time they correct and define variations in the normal sounds and the significance of adventitious sounds.

Methods of Examination.—Both the mediate and the immediate methods of auscultation are employed, each having its proper field.

By mediate auscultation, with the ear applied directly to the chest, the observer hears not only the sounds of the heart, but he also notes the impulse and other tactile sensations, and is thus able to gauge more accurately the period of the cardiac cycle at which both normal and abnormal sounds occur.

Immediate auscultation, employing a stethoscope, in addition to the advantages noted under Auscultation of the Lung, is especially valuable in the examination of the heart, as it *(a)* limits the sound heard to that portion of the chest wall covered by the thoracic end of the stethoscope. This is very important, as the cardiac orifices are situated close to each other, and it is necessary to determine where both normal and adventitious sounds are heard with the greatest intensity; while with the ear alone we are apt to include more than one valve area in the scope of the examination. *(b)* It cuts off external sounds. *(c)* It intensifies some of the sounds produced in the heart, especiallv those due to valve action and murmurs.

In selecting a stethoscope, the following points should be observed:

The binaural forms are mostly used. The advantages of the rigid ones over those made with flexible tubes are increased power of conduction and conveyance of cardiac impulse, as well as sound. The disadvantages are a certain resonating effect, which increases the intensity and may change the character of the higher-pitched valve sounds and murmurs. In order that the stethoscope be a good conductor of sound, whether rigid or made of flexible (rubber) parts, the interior should be absolutely smooth. The thoracic end of the stethoscope should be small, so as to fit closely to the surface of the chest and also limit the field of examination to a small area. The ear pieces should fit the ear without producing pain or discomfort. Usually too small ear pieces are selected, which penetrate too deeply into the canal. The ear pieces should merely close up the wider portion of the orifice. The spring holding the ear pieces in place should not be too stiff, and where the instrument has to be used for long periods of time each day a form of stethoscope should be used in which the pressure of the spring can be regulated.

The examination should be made methodically, and those portions of the heart should be first examined where the cardiac sounds are most distinct, both as to intensity and their relation to the ventricular systole.

Normal Cardiac Sounds.—Two sounds are produced by cardiac action, each having a definite relation to the phase of cardiac cycle and differing in time, character, position of maximum intensity and area of diffusion.

First Sound.—The first sound of the heart corresponds to the ventricular systole, closure of the mitral and tricuspid valves and opening of the aortic and pulmonary valves, with forcing of the blood stream from the right and left ventricular cavities into the pulmonary artery and aorta. The first sound of the heart is a prolonged, low-pitched, dull sound, which has some resemblance to the sound made in the pronunciation of the syllable "lubb."

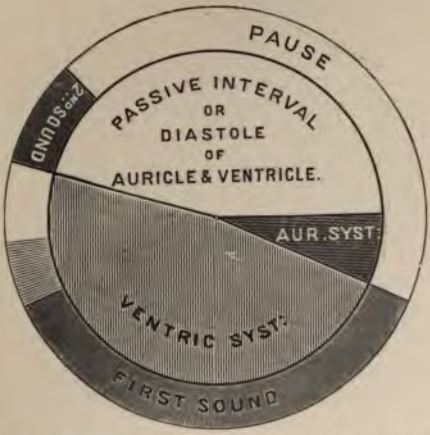
The cause of the first sound of the heart is still a matter of dispute. Numerous theories have been advanced, but no one of them has been universally adopted. The consensus of opinion is that the first sound of the heart is composed of two elements: *(a)* The muscular element, due to the contraction of the cardiac muscle and its impact on the chest wall, giving the prolonged, dull, low-pitched quality. *(b)* The valve element, due to the closure of the mitral and tricuspid valves and stretching of the chordæ tendineæ and papillary muscle, causes the short, sharp and high-pitched quality. As will be explained under Modifications of Normal Sounds, the first sound will vary according as one or the other of these elements predominates.

The first sound of the heart is heard most distinctly at the region of the apex. At this point both muscular and valve elements are present. The muscular element predominates, because at the time it is heard the rigidly-contracting apex of the heart strikes the chest wall and is momentarily held in contact with it, conveying the vibrations to the thorax.

Second Sound.—The second sound of the heart corresponds to the closure of the aortic and pulmonary valves. It is shorter, sharper and higher pitched than the first sound of the heart, being somewhat similar to the sound produced by the pronunciation of the syllable "dubb." It is composed of one element only, the valve sound, which is short, tense and snapping, and may be "very closely imitated by stretching with more or less suddenness a piece of cloth or a moistened membrane." (Sanson.)

The second sound of the heart, being composed entirely of the sounds made by the closure of the aortic and pulmonary valves, is heard most distinctly over the base of the heart.

FIG. 44.



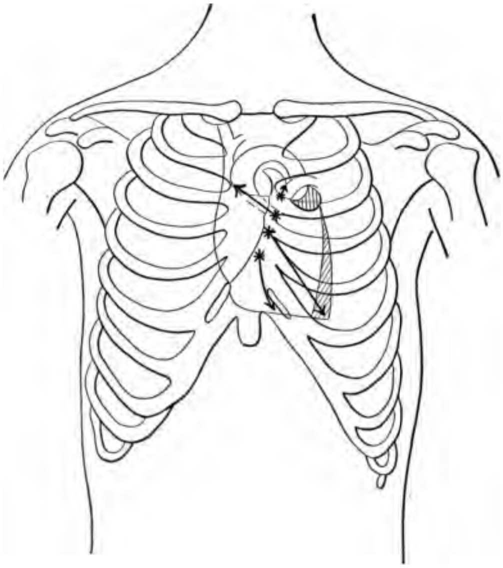
Diagrammatic representation of the movements and sounds of the heart. (After SHARPEY.) This diagram shows merely the general relations of the several events, and does not represent exact measurements.

In a heart beating seventy-two times a minute, Foster estimates each entire cardiac cycle as occupying about 0.8 sec., of which 0.3 sec. represents the duration of the systole of the ventricle, 0.4 sec. the diastole of both auricle and ventricle, or the "passive interval," and 0.1 sec. the systole of the auricle.

Only one "pause" is marked here—sometimes called the "long pause;" some writers describe a "short pause" also—indicated in the diagram by the small space between the first and the second sound.

Valve Areas.—Listening over certain portions of the chest, the first and second sounds are heard with varying intensity and clearness, and it is possible to appreciate the sounds made at the different valves. The areas at which the valve sounds are best heard are called valve areas, and are named according to the valve whence the sound proceeds—aortic area, pulmonary area, tricuspid area and mitral area. The areas do not correspond to the anatomical situation of the valves, but to points on the surface of the chest, to which the vibrations made at the valves are conducted with the greatest intensity. (Fig. 45.)

FIG. 45.



From above downwards, showing the anatomical positions of the pulmonic, aortic, mitral and tricuspid valves. The arrow-heads show their respective sites of audibility. Shaded portion shows part of left heart projecting beyond the right heart.

The Mitral Area.—The valvular element of the first sound, heard at the fifth intercostal space, is that produced by the mitral valve. The location of the mitral area does not correspond with the anatomical site of this valve, which is situated behind the left half of the sternum, opposite the insertion of the third left costal cartilage. The conduction of the vibrations made at the mitral valve to the apex is through the tense chordæ tendineæ and papillary muscle, and the ventricular wall.

The Tricuspid Area.—The sound made at the tricuspid valve is heard over the lower end of the sternum. Conduction of the sound from seat of production to the lower end of the sternum is due to conveyance by the chordæ tendineæ and papillary muscle on the right side to that portion of the right ventricular wall which is in close contact with the lower left edge of the sternum.

The Aortic Area is in the second right intercostal space, close to the sternum, and the second right cartilage has been called the "aortic cartilage." The aortic valve is situated lower than this area, being behind the left half of the sternum to the left of the right third interspace. The vibrations made at the aortic orifice are conveyed to the aortic area through the tense walls of the aorta, which at this point come closely in contact with the chest wall.

The Pulmonic Area is at the second left intercostal space, close to the sternum, and corresponds more nearly than do the other areas to the anatomical site of the valve, which is slightly lower under the third intercostal cartilage, extending slightly into the second interspace.

Modifications of the Cardiac Sounds.—The cardiac sounds may be modified *(a)* in intensity, which may be increased or diminished, and *(b)* in rhythm, which concerns the duration of the pause which separates the sounds.

(a) CHANGES IN INTENSITY.—This may involve both sounds in nearly equal proportions, or it may affect only one sound. Normally, the intensity of the first and second sounds of the heart bear a definite relation to each other. The intensity of the sounds may be relatively increased or diminished without indicating any pathological change in the heart, as the intensity is largely dependent upon the degree of endocardial and blood pressure. The increase or diminution affecting

both sounds should always be considered in connection with the tension and compressibility of the pulse.

The cardiac sounds vary normally within certain limits. In the young the first sound is shorter, higher pitched and more ringing in character, with a greater prominence of the valvular element than in the adult; while the second sound, heard over the pulmonic area, is louder than the aortic. After middle life the first sound is duller, more prolonged and lower pitched, the mitral and tricuspid elements being relatively less prominent, while the aortic sound becomes more accentuated and is louder than that heard in the pulmonic area.

In adult life the first sound varies greatly. "In thin, nervous people the first sound is high pitched, short and more ringing in character. In the robust and vigorous it is low pitched, prolonged and rumbling, while in the fat and indolent it is indistinct and short."

The cardiac sounds are modified (1) by those conditions which increase or diminish the conduction of the sounds to the surface, as *(a)* thickness or thinness of the soft parts, *(b)* condition of the bony thorax, *(c)* changes in the lung and pleura, *(d)* changes in the pericardium; (2) by changes in the circulatory system.

INCREASE IN INTENSITY.—Both sounds of the heart are increased in intensity *(a)* when the thorax is thinly covered by soft parts; *(b)* when the bony thorax is flexible and readily thrown into vibration; *(c)* when, through changes in the pleura or lungs, the heart is uncovered to a greater extent than normal; *(d)* when the pulmonary tissue that normally covers the heart is rendered more homogeneous by consolidation and the cardiac sounds are conveyed to the surface with a peculiar distinctness, the valvular element being especially prominent. Cavities with tense walls and pneumo-thorax also cause increased conduction of the normal heart sounds. *(e)* Adherent pericardium causes increase in the cardiac sounds, due both to increase of power of conduction and to secondary changes induced in the heart muscle.

Increase in intensity may be due to conditions of the circulatory system.

First Sound.—The intensity of the first sound may be increased both in the muscular and valvular elements or only in one. In cardiac overaction, and associated with low tension in the blood-vessels, as occurs in fevers, physical exertion, mental and emotional excitement and in Grave's disease, the first sound is high pitched and short, and the valve element is most marked. When cardiac hypertrophy is present (compensating), and when cardiac overaction is associated with high tension in the blood-vessels, the muscular element predominates and the sound is dull, prolonged and booming. When cardiac hypertrophy ceases to be compensating, the first sound, although more intense than normal, becomes short, sharp and "flapping," the valvular element being most pronounced.

Second Sound.—Increase in intensity of the second sound of the heart may be due to the accentuation of both aortic and pulmonic sounds, or it may be limited to one sound.

Accentuation of the Aortic Sound.—Accentuation of the aortic sound is usually dependent upon increased pressure within the aorta, which causes the valves to close with greater tension and suddenness than normally. The increased pressure within the aorta may be *transient,* as when due to increased cardiac action or to obstruction to the flow of blood from the arteries and capillaries, as spasm from chilling of the surface, nervous shocks, etc.; or it may be *permanent,* as occurs in cardiac hypertrophy with dilatation and in arterial sclerosis, kidney disease, etc. The aortic second sound is usually accentuated in pregnancy, because of the marked increase in blood mass. Accentuation of the aortic second sound is usually associated with a corresponding increase in the intensity of the first sound.

This relation is frequently disturbed by the muscular power of the heart becoming insufficient, when the second sound becomes accentuated and the first sound grows progressively weaker and feebler. This association is always a grave symptom.

As the aortic sound is normally weaker than the pulmonic sound until middle adult life, the intensity of these two sounds must be compared in order to detect changes in the normal relative intensity. After middle adult life, normally, the aortic sound approaches more nearly that of the pulmonic, and the latter becomes more intense.

Accentuation of the Pulmonic Sound.—The sound over the pulmonic area is accentuated by conditions which raise the blood pressure in the pulmonary artery, as acute and chronic congestion of the lungs, acute inflammatory diseases, anæmia, and obstructive diseases of the lungs (emphysema, fibroid induration). As will be shown later, increased tension in the pulmonary artery is associated with corresponding hypertrophy of the right ventricle. When endo-ventricular pressure in the right ventricle is raised above a certain point, insufficiency of the tricuspid valve occurs (safety-valve action), and there ensues a corresponding diminution in the accentuation of the pulmonic sound.

Diminution in Intensity.—The cardiac sounds are diminished in intensity by (1) those conditions which interfere with conduction to the surface, as *(a)* when the thorax is thickly covered with adipose tissue; *(b)* when the bony thorax is rigid; *(c)* when the heart is separated from the chest wall by distended lung tissue (emphysema) or displaced by effusions into the pleural cavity; *(d)* by effusions into the pericardium and pneumo-pericardium. In these conditions the character of the sounds is *muffled* (2) by changes in the heart itself.

First Sound.—The intensity of the first sound of the heart is diminished by all conditions of muscular feebleness, both relative, as when hypertrophy is no longer compensating, and absolute, as degenerations of the cardiac muscle. The sound in these conditions is short, sharp, and the booming character is lacking and replaced by a peculiar "flapping" quality, dependent upon the valve element.

The Second Sound.—The aortic sound is diminished in intensity by (1) conditions which cause low blood pressure, decrease in the aortic recoil, and due to *(a)* feeble heart action; *(b)* imperfect or slow filling of the aorta, as occurs in aortic stenosis, mitral stenosis and mitral regurgitation; *(c)* loss of blood mass from hemorrhage, diarrhœa, etc., *(d)* or increased rapidity of flow from the arteries and capillaries. (2) Changes in the valves themselves, diminishing their elasticity, as in endocarditis.

The pulmonic sound varies within much narrower limits than does the aortic, as the pressure within the pulmonary artery is more uniform. Marked diminution in the pulmonic second sound is generally indicative of failure of the right ventricle.

CHANGES IN RHYTHM.—The rhythm of the heart may be disturbed by alterations in (1) the *relative time of the pause.* When the heart beat is increased in frequency, in proportion to the rate of the heart, there is a corresponding shortening of the pause between the second and first sounds of the heart (diastole), but this is never sufficiently marked to disturb the normal rhythm.

(2) In embryo-cardia, in which the two sounds follow each other at equal intervals, giving the peculiar tick-tack character of the fœtal heart. This condition is generally indicative of profound muscular weakness, and occurs in the terminal stage of cardiac disease and impending cardiac failure in typhoid and other acute diseases. It is an especially grave symptom in typhoid and in Grave's disease.

(3) Canter or Gallop Rhythm.—In this condition

three sounds of the heart are noted. The three sounds may be equally distinct, or the added sound may be of a different character and may occur at any period of the cardiac cycle. The rhythm is similar to that of the footfalls of a galloping horse, hence the name, "gallop rhythm," or "bruit de galop."

(4) Reduplication of Sounds.—Both the first and second sound may be reduplicated or doubled. A slight degree of reduplication of the second sound is physiological, and can be induced by holding the breath. In this condition the temporary heightening of the pressure in the pulmonary artery causes tardy closure of the pulmonic valves. Doubling of the first sound may also occur, but is usually more apparent than real. To account for the reduplication of the sounds, many theories have been advanced. As the reduplication seems to concern chiefly the valvular elements of the sounds, it is evidently due to the fact that the time of the maximum intensity of the sounds made at the different valves on one side of the heart does not correspond to that of their fellows on the other side of the heart, being separated by an appreciable interval.

Abnormal or Adventitious Sounds. Murmurs. — The abnormal or adventitious sounds heard over the præcordia may be produced (1) within the heart (endocardial), or (2) in the pericardium, lung or pleura (exocardial).

Endocardial Murmurs.—Endocardial murmurs are due to vibrations made at the valve orifices by the movement of the blood through them. Normally, the blood passes the valvular orifices without any sound being produced except the normal first and second sounds. This noiseless passage of the blood demands (1) normal anatomical conditions at the openings, as far as smoothness of surface, relation of the size of the opening, both relative and absolute, to the part beyond, and perfect closure of the opening by the valves during the normal period. (2) A certain specific gravity of the blood, and (3) a definite rapidity and force of the current. Disturbance of any one of these factors may be sufficient to break up the moving column of blood into "fluid veins" whose vibrations are rapid enough to produce a sound or *murmur* when transmitted to the surface of the body.

Classification of the Murmurs.—Murmurs are classified as (1) organic or valvular, (2) inorganic or non-valvular.

ORGANIC OR VALVULAR MURMURS.—Fluid veins are made whenever the blood is forced through a narrow portion of the circulatory tract into a part that is larger. In the normal heart the relative sizes of the opening and cavities of the heart and aorta are such that fluid veins are not formed under usual conditions.

The necessary physical condition for the production of fluid veins may be produced by anatomical changes at the valve orifices which *(a)* interfere with the passage of blood in normal direction through the opening, as narrowing of opening or roughing of the surface (obstruction or stenosis); *(b)* which allow of abnormal or backward flow (regurgitation), due to imperfect closure of opening, from change in valves (absolute insufficiency), or to dilatation of opening (relative insufficiency). *(c)* At the aortic and pulmonary orifices, increase in the size of the vessel may so change the relation between the size of the opening and the cavity beyond as to favor the production of fluid veins.

INORGANIC OR NON-VALVULAR MURMURS.—These may occur with normal cavities, orifices, and are due to *(a) changes in the blood, lowering its specific gravity.* This allows the fluid veins to be more readily formed in both normal and pathological conditions of the valves and orifices, and also in the great vessels. The specific gravity of the blood is lowered in anæmia, hydræmia, and the murmurs are known as *anæmic* or *hæmic.*

(b) Increase in rapidity and force of the current of blood. It is possible for the action of the heart to be so energetic and rapid as to produce vibrations

(inorganic murmurs) at the different orifices when they are normal in size and structure and the specific gravity of the blood is unchanged. The murmurs are known as *functional* or *dynamic*.

A certain amount of force is also necessary to produce murmurs in pathological conditions of the heart.

In every case of endocardial murmur one or more of the above factors are present in varying degrees, and each has an influence in producing certain characteristic features of the murmur.

Characteristics of Murmurs.—The recognition of an endocardial murmur and the determination of its origin and pathological significance depend upon its (1) quality, (2) intensity, (3) time or rhythm, (4) point of maximum intensity, (5) area of diffusion, (6) duration, (7) permanency and influence of exertion and posture.

QUALITY OF THE MURMUR.—Endocardial murmurs vary widely in relation to their quality, and it is sometimes difficult to determine whether or not a slight variation from the normal sound is due to the presence of a murmur. All murmurs have in a greater or less degree a liquid character. In addition to this, they may have a peculiar rushing or harsh, blowing sound, which may be imitated by the voice in pronouncing the syllables *uf*, *uv*, *us*, *ush*, *uz*; or they may have a soft, blowing sound, as represented by the syllables with vowel sounds of *oo*, *u*, *ah*, *au*; or, again, they may have a rougher, more vibrant character, as in *uur*, *orr*, *arr*. At times the vibrations may have a musical sound.

The quality of the sound does not determine whether the murmur is organic or inorganic, although hæmic murmurs as a rule are of the soft, blowing character.

The quality of the murmur may change from time to time in the individual case, depending upon the changes in size and structure of the orifice and valves, the relative size and condition of the space into which the blood is forced, the varying force of the cardiac action, and the presence or absence of anæmia.

INTENSITY OF MURMURS.—The intensity or loudness of endocardial murmurs shows great variability in lesions involving the different orifices or in the same orifice at different times.

The intensity of a murmur depends upon *(a)* the force of the blood current, *(b)* the size and shape of the orifice at which the murmur is made and the state of the cavity into which the vibrating blood is thrown, and *(c)* the condition of the blood.

Systolic murmurs, the force being supplied by the ventricular contraction, are usually louder, other things being equal, than diastolic murmurs, whose force is due to the elastic recoil of the aorta and pulmonary artery. In pre-systolic murmurs the force is supplied by the auricle.

The varying degrees of intensity are of important diagnostic and prognostic value, as diminution in the intensity is frequently the first sign of failing cardiac power. On the contrary, gradual increase in the intensity of the murmur may indicate gradual restoration of compensation.

The size and shape of the orifice influences the intensity of the murmur according to the character of the vibrations produced. The narrower the orifice in proportion to the size of the cavity beyond, the more intense is the murmur as a rule.

The intensity of the murmur is also influenced by the character of the circulating blood. When the specific gravity is reduced by diminution of the corpuscular elements or by the decrease in proteids (hydræmia), the murmurs are louder and their quality is changed. As the specific gravity blood becomes more nearly normal, the loudness of the murmurs decreases.

The intensity of the murmurs made at the different orifices of the heart varies considerably with the posture of the patient. Of murmurs made at the mitral and tricuspid orifices, the obstructive murmurs are loudest when the patient is sitting or standing, and become feebler or disappear when he is lying down. Regurgitant murmurs at these orifices, on the other hand, are most distinct when the patient is lying flat on the back, and become more indistinct as he resumes the upright posture. Murmurs made at the aortic and pulmonary valves are not influenced to the same extent by posture. Aortic regurgitation is loudest when the patient is in the recumbent position, and is still further increased if one or both arms are raised above the head. Systolic anæmic murmurs heard over the base of the heart are loudest when in the recumbent position, and their intensity is also increased at the end of expiration and just at the beginning of inspiration.

TIME OR RHYTHM.—As endocardial murmurs may be produced at any part of the cardiac cycle, the most important characteristic is its relation to the normal cardiac sounds. As the ventricular systole is the most prominent phase of the cardiac cycle, being marked by the visible apex beat, the carotid pulse and the first sound of the heart, murmurs are timed by it. Murmurs are described as systolic and diastolic. On account of the relative length of the diastolic pause, those murmurs that are only heard during that portion of diastole that immediately precedes the occurrence of the first sound have been named pre-systolic murmurs.

SYSTOLIC MURMURS may be (1) obstructive, as when produced by blood flowing in a normal direction through valves that should be open at this period of the cardiac cycle—that is, the aortic and pulmonic valves—or (2) regurgitant, as when the blood is forced back through valves that should be closed, namely, the mitral and tricuspid valves.

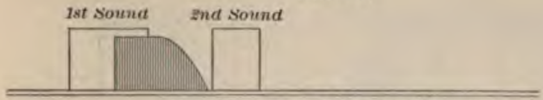
DIASYSTOLIC MURMURS are (1) regurgitant, being due to leakage at either the aortic or pulmonary valves, which should normally be closed at the completion of systole and occurrence of the second sound.

(2) PRE-SYSTOLIC MURMURS are obstructive, being produced at the mitral and tricuspid valves during the

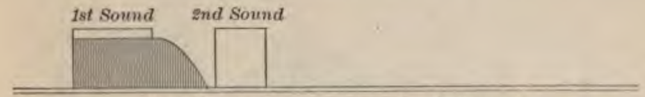
auricular systole, which immediately precedes the ventricular systole and the occurrence of the first sound.

As the time at which the murmur occurs and its relation to the normal sound is most important for diagnosis, it is necessary that it be correctly determined. Ordinarily the first sound of the heart is the more easily detected than the second sound, and is used to time the murmur by auscultation. But whenever it is masked by the occurrence of a systolic murmur, the time of the different phases of the cardiac cycle should be determined by listening over the valve areas, where second sounds are heard with greatest distinctness. With these facts well impressed, the time of the abnormal sound can be fixed most readily.

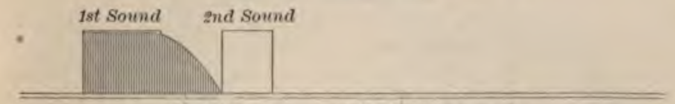
FIG. 46.



Systolic murmur, following first sound.



Systolic murmur, accompanying first sound.

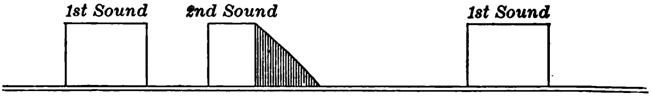


Systolic murmur, replacing first sound.

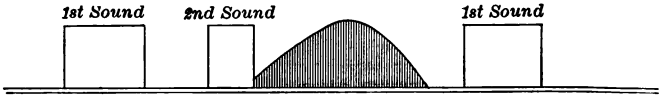
The differential stethoscope is frequently an aid in determining the time of a murmur, as it allows of comparison, through the two chest pieces placed at different portions of the chest, of the murmur with the first or second sound heard at an area uninfluenced by it.

Murmurs may accompany or take the place of the normal heart sounds, and their duration is also measured by their relation to the normal cardiac periods. As the first sound of the heart is made up of both muscular and valvular elements, systolic murmurs will influence one or the other of these elements by accompanying it or replacing it, according to the changes that have occurred at the orifice and the effect of these lesions on the ventricular wall and cavity. (Fig. 46.) Diastolic murmurs may accompany, take the place of, or follow the second sound; and the presence or absence of the valvular sound, and whether or not the murmur follows it, determine to a certain extent the condition of the orifice of the valves themselves. (Fig. 47.)

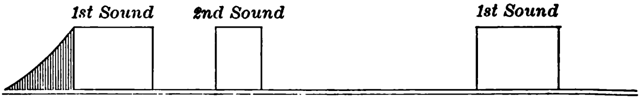
FIG. 47.



Diastolic murmur, following second sound.



Late diastolic murmur.

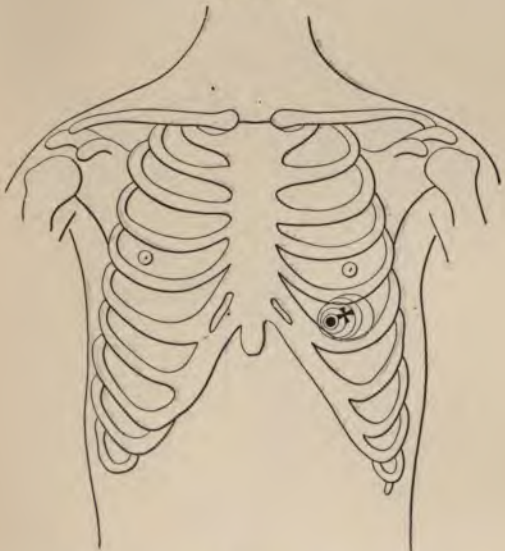


Pre-systolic murmurs.

POINT OF MAXIMUM INTENSITY AND AREA OF DIFFUSION.—The point at which a murmur is most distinctly heard determines the orifice at which it is made.

The points of maximum intensity of the endocardial murmurs do not correspond to the anatomical site of the valves nor entirely to the valve areas. The conduction of murmurs to their points of maximum intensity is dependent *(a)* on the direction of the blood current which produces the murmur, and *(b)* on the readiness with which certain portions of the heart, by reason of their anatomical relations, transmit the vibrations made at the point of obstruction or regurgitation to the surface. From the point of maximum intensity, the murmur is transmitted in different directions (area of diffusion). The extent and direction of the transmission are variable, chiefly according to *(a)* the strength and character of the vibration producing the murmur; *(b)* the method of primary conduction to the surface, whether mainly by blood current or solid structures (heart, lung, etc.), and *(c)* to the diffusion over the chest from the point of maximum intensity by the bony thorax and soft parts. It is necessary to consider the point of the maximum intensity and area of diffusion of the murmurs made at the different orifices.

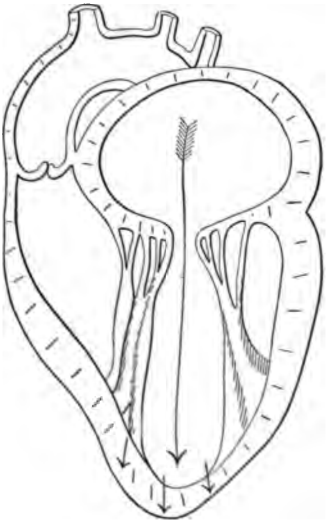
FIG. 48.



Mitral pre-systolic murmur. ✠ Apex of heart.

Murmurs Made at the Mitral Orifice. MITRAL PRESYSTOLIC MURMURS.—The point of maximum intensity is slightly to the right of the apex of the heart (left ventricle), whether it is in the normal position or displaced, and its area of diffusion is, as a rule, very slightly beyond the region of the apex. (Fig. 48.) The localization of a murmur at the region of the apex of the left ventricle is due *(a)* to the direction of the vibrating blood current, which is concentrated at the apex by the valves of the narrowed orifice. *(b)* The flaccid condition of the overlying right ventricle prevents the transmission of the vibrations to the surface in that direction. *(c)* The left ventricle being also in a state of relaxation and at its greatest distance from the chest wall, the vibrations, conducted to the surface through the overlying lung, influence a very small area of chest. (Fig. 49.)

FIG. 49.

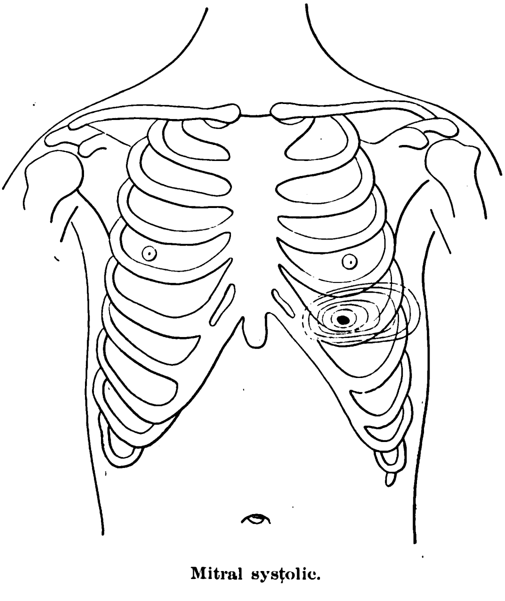


Mitral obstruction. Large arrow direction of current of blood.

A wider area of diffusion of the pre-systolic mitral murmur is generally due either to unusually close relation of the apex of the left ventricle to the chest wall, or to increased power of conduction on the part of the tissues surrounding the apex of the heart, or to the character of the vibrations.

The pre-systolic murmur heard at the apex is generally due to obstruction at the mitral orifice, although murmurs with this rhythm and localization without mitral obstruction have been heard in aortic regurgitation (Flint murmur), in aortic obstruction and with adherent pericardium.

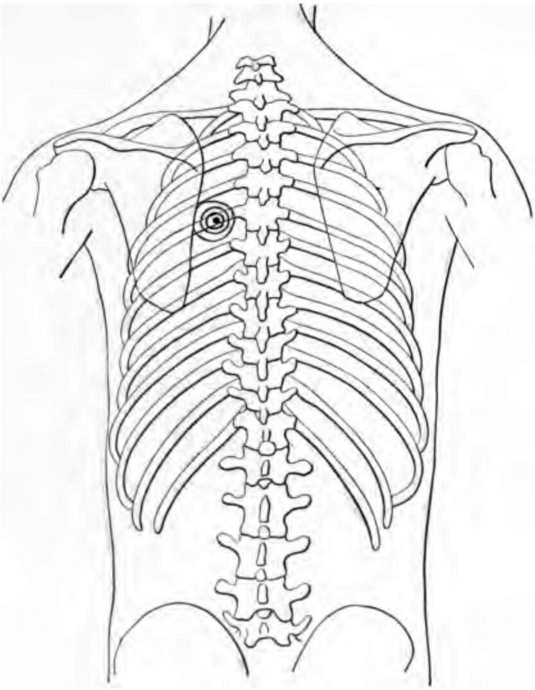
FIG. 50.



Mitral systolic.

MITRAL SYSTOLIC MURMURS.—The point of maximum intensity is at or close to the apex. The area of diffusion extends also around this point, and to the left beyond the præcordia into the axillary region. (Fig. 50.) The murmur is also heard in the back, the point of maximum intensity being in the left interscapular space, a little above the angle of the scapula, opposite the spines of the fifth and sixth dorsal vertebræ. (Fig. 51.) The area of diffusion is usually limited to the left scapular and interscapular regions.

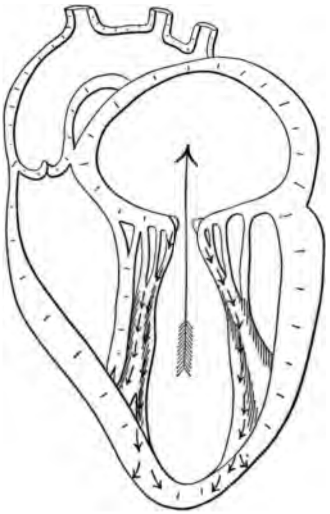
FIG. 51.



Mitral systolic.

The murmur made at the mitral valve (situated behind the left half of the sternum at the level of the fourth interspace) reaches the surface in front over the apex of the heart and behind in the scapular region by two different lines of conduction. (Fig. 52.) Conduction to the apex is mainly by the tissues of the left ventricle, which at the time of the first sound of the heart are in a state of extreme tension. The vibrations made by the regurgitant stream are transmitted *(a)* by the leaflets of the mitral valve through the tense chordæ tendineæ and papillary muscle to the apex of the left ventricle. *(b)* The apex of the left ventricle at this time is in direct contact with the chest wall, to which it transmits the vibrations of the murmur. *(c)* From the point of contact (the point of maximum intensity) the vibrations are conveyed along the ribs in all directions (diffusion). Over the præcordia they are deadened by the right ventricle, so that their diffusion to the right

FIG. 52.

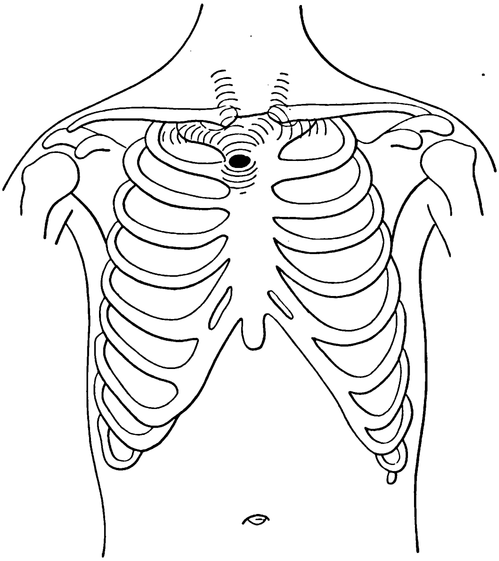


Mitral regurgitation. Large arrow shows direction of current of blood. Small arrows show conduction of vibration to the apex by chordæ tendineæ and papillary muscles.

and upwards is limited. The vibrations are conveyed by the ribs to the axillary region. The murmur is heard over wider area when the left ventricle is much hypertrophied, and the apex is displaced downward and to the left, and is in contact with the chest wall to a greater extent.

Conduction of the mitral regurgitant murmur to the scapular region is from the left auricle, which is thrown into vibration by the stream of regurgitant blood. From the auricle the vibrations are transmitted through the overlying tissues to the surface. The mitral regurgitant murmur heard in the back corresponds more nearly to the anatomical site of the valve.

FIG. 53.

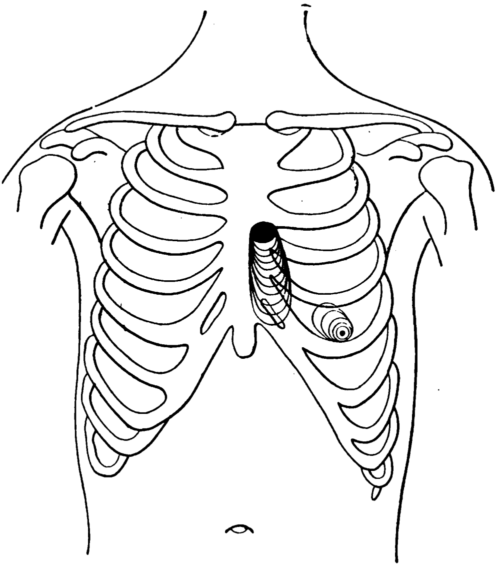


Aortic obstruction.

Mitral systolic murmurs may occur in mitral insufficiency, due to acute or chronic endocarditis, causing deformity of the leaflets, dilatation of the left ventricle, or to dilatation of the left ventricle enlarging the mitral orifice and causing relative insufficiency of the leaflets to close the opening.

Murmurs Made at the Aortic Orifice. SYSTOLIC MURMURS.—The point of maximum intensity as a general rule is situated over the manubrium sterni, and not at the second right cartilage (aortic area). The area of diffusion is upward into the carotids, and laterally along the clavicles and over the subclavian arteries. Conduction is by the current of blood in aorta to the point of maximum intensity. The vibrations are transmitted through the walls of the aorta to the manubrium sterni and clavicles. (Fig. 53.)

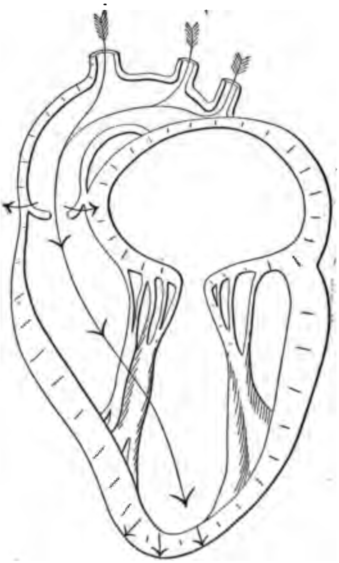
FIG. 54.



Aortic regurgitation.

Systolic murmurs in the aortic region may be caused by conditions that lead to the production of vibrations at the valve orifice itself or in the aorta just beyond. *At the orifice:* (1) Narrowing of the lumen of the opening by changes in the valves (stenosis). (2) Roughening of the surfaces by vegetations, calcareous deposits without diminution in the size of the orifice (non-stenotic). *In the aorta:* (1) Roughening of the inner walls of the aorta. (2) Dilatation of the aorta.

FIG. 55.



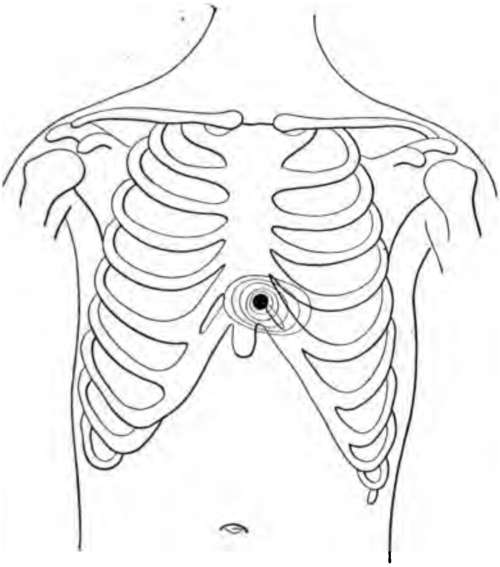
Aortic regurgitation. Arrows show lines of conduction of vibrations.

Inorganic causes: (1) Functional or dynamic, due to increase of cardiac force, as in cardiac overaction caused by mental or emotional excitement, physical exertion, Grave's disease. (2) Hæmic or anæmic.

Murmurs due to anæmia are heard in the aortic area or over the sternum, but are transmitted into the vessels of the neck with increasing intensity.

Aortic Diastolic Murmurs.—The point of maximum intensity is not, as usually described, at the second right intercostal cartilage, but over the sternum and at the left edge, opposite the third interspace or fourth costal cartilage. The area of diffusion is downward along the sternum, and also to the apex of the left ventricle. (Fig. 54.) Conduction of the aortic regurgitant murmur to the sternum at the point of maximum intensity is due to the intimate relation of the aortic cusps with the auriculo-ventricular septum of the right side of the heart. The vibrations made by the regurgitant stream are transmitted from the aortic valves through the right auriculo-ventricular septum to that portion of the sternum that is in closest contact with it, viz.: from the third left interspace to the sixth rib on

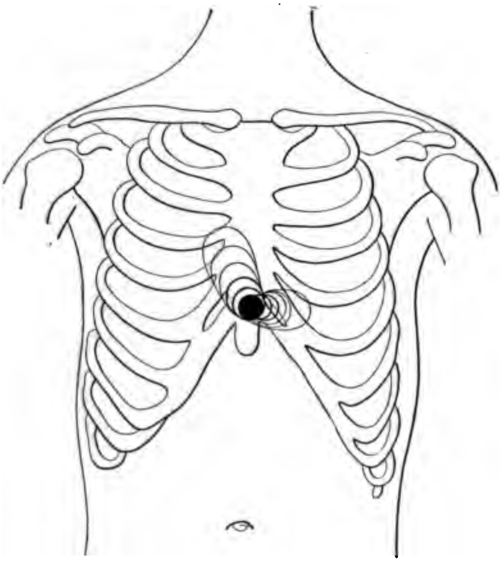
FIG. 56.



Tricuspid obstruction.

the right side. Conduction of the murmur to the apex of the left ventricle is by the regurgitant stream. (Fig. 55.) The character of the murmur heard at the apex may differ greatly from that present over the sternum. Variations in the location of the point of maximum intensity and area of diffusion are dependent upon the cause and the nature of the secondary changes in the heart and aorta (see Aortic Regurgitation).

FIG. 57.



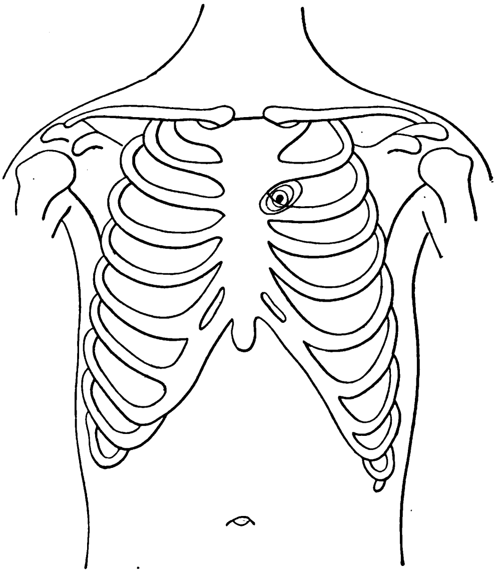
Tricuspid regurgitant.

Regurgitation at the aortic orifice may depend upon *(a)* absolute insufficiency of the valves, due to vegetations, loss of substance, thickening and distortion of the valves, congenital malformation (rare); *(b)* upon rupture of the valves; *(c)* upon relative insufficiency, as when the valves are normal, but insufficient to close the orifice on account of dilatation of the aortic ring.

Murmurs Made at the Tricuspid Valve.—Tricuspid murmurs are usually most distinct over the lower portion of the sternum and along the lower left costal cartilages.

Tricuspid Pre-systolic Murmurs.—The point of maximum intensity is over the lower portion of the sternum, at the upper edge of the ensiform cartilage, and the area of diffusion corresponds with the area of superficial cardiac dullness. (Fig. 56.) Conduction to the surface of the vibrations made at the tricuspid valve is from that portion of the right ventricle which is in direct contact with the sternum.

Fig. 58.

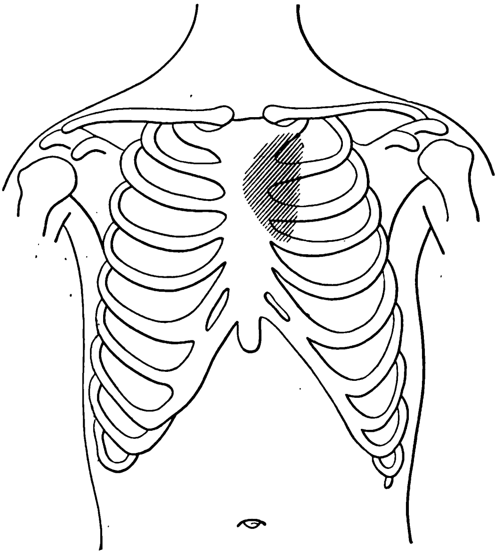


Pulmonic systolic—stenotic.

The tricuspid pre-systolic murmur is extremely rare, and, when detected, is diagnostic of obstruction at the orifice, either congenital or acquired.

TRICUSPID SYSTOLIC MURMURS.—The point of maximum intensity is at the base of the ensiform cartilage, and the area of diffusion from the point of maximum intensity is upward and to the right, not above the third rib, and to the left toward the apex of the heart. (Fig. 57.) Conduction of the murmur in these two directions is by the regurgitant stream, and by transmission through chordæ tendineæ to apex, the same as in mitral regurgitation.

FIG. 59.

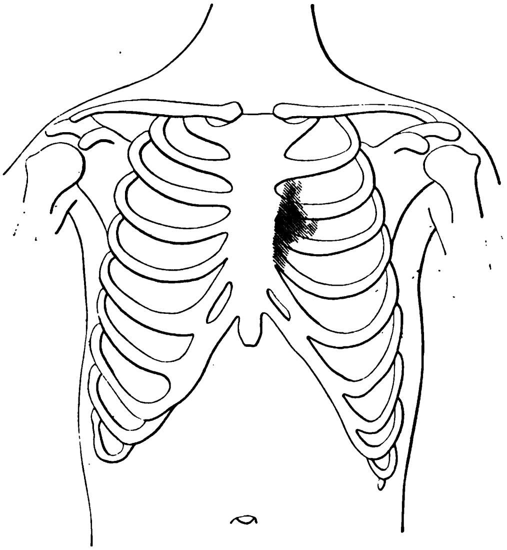


Pulmonic systolic anæmic murmur.

Vibrations made at the valve are carried by the regurgitant current of blood upward into the auricle over which the murmur is heard. The vibrations are transmitted to the right ventricular wall by the attachment of the valve and by the tense chordæ tendineæ and papillary muscle. The point of maximum intensity and the area of diffusion vary according to the degree of hypertrophy and dilatation. At times the murmur corresponds in location to the mitral area. It is rarely heard beyond the præcordia, and is not carried into the axilla nor heard behind in the scapular region.

Regurgitation at the tricuspid orifice results from insufficiency due to *(a)* the effects of acute and chronic endocarditis, to vegetations, to induration or distortion of the valves, and to shortening of the chordæ tendineæ; *(b)* from dilatation of the right ventricle and tricuspid ring, with relative insufficiency. The dilatation may be permanent or transient (safety-valve action). (See **Regurgitation at Tricuspid Orifice.**)

FIG. 60.

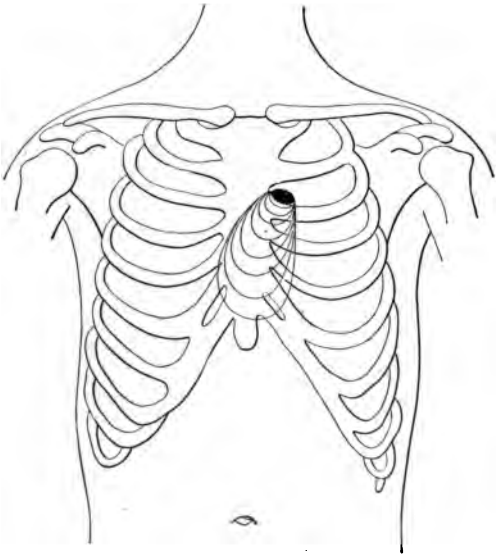


Anæmia, pulmonic systolic.

Murmurs at the Pulmonary Orifice. PULMONIC SYSTOLIC MURMURS.—The point of maximum intensity is at the left intercostal space, close to the sternum. The area of diffusion may be circumscribed around the point of maximum intensity, or may extend upward along the course of the pulmonary artery and laterally to the left. (Fig. 58.)

The occurrence of a systolic murmur at the pulmonic area is extremely common, and may be due *(a)* to organic changes in the valves, producing obstruction (rare), or *(b)* to narrowing of the lumen of the pulmonary artery by changes in the surrounding structures or torsion from displacement of the heart; *(c)* to anæmia, either alone or associated with dilatation of the

FIG. 61.



Pulmonic diastolic.

artery (most commonly). In anæmic murmurs the point of maximum intensity and area of diffusion is more variable than in those dependent on stenosis at orifice. (Figs. 59 and 60.)

PULMONIC DIASTOLIC MURMURS.—This murmur is very rare. The point of maximum intensity is over the pulmonic valve area. The area of diffusion is along the left edge of the sternum. It may occur *(a)* from congenital malformation or ulcerative endocarditis; *(b)* from relative insufficiency caused by dilatation of the pulmonary artery or orifice. (Fig. 61.)

Exocardial Murmurs. A. ARTERIAL MURMURS.—The presence over the aorta of the normal cardiac sounds, and of murmurs made at the aortic orifice and in aorta, has already been mentioned, and will be still further considered under diseases of the aorta.

When the stethoscope is lightly applied to the carotid and subclavian arteries, the normal first and second cardiac sounds may be faintly heard; the first sound weak and low pitched, the second sound louder, higher pitched, and snapping, corresponding to its character at the aortic cartilage. Over the arteries that are further removed from the heart, as the brachial, crural, femoral, etc., no sound is normally heard *unless the pressure of the stethoscope is sufficiently hard to compress the artery* when there is heard a systolic murmur ("pressure murmur"), whose intensity corresponds to the degree of narrowing, and which may have a musical quality.

Murmurs heard over the arteries may be produced *(a)* at the cardiac orifice or aorta, and conducted into them, or *(b)* in the blood-vessels at the point where heard.

SYSTOLIC MURMURS made in the arteries may be due *(a)* to roughening of the inner coat; *(b)* to narrowing of the lumen, which may be caused by changes in the wall of the artery, as calcareous plates, or due to pressure from surrounding structures, as lymph nodes, etc.;

(c) to dilatation of the vessel from fevers, acute local inflammation, chronic dilatation and vaso-motor conditions, causing paresis of the vessel itself at certain points; *(d)* to anæmia. Two or more of these causes may be combined in any given case. The association of localized vaso-motor dilatation with anæmia is particularly common.

Over the subclavian arteries a short, blowing, systolic murmur ("whiff") is normally heard when the chest is fully expanded. This murmur may become constant and harsher if the artery is contracted or a sharp bend produced by retraction of the apex of the lung or pleural adhesions. This subclavian murmur is frequently mistaken for an aneurismal murmur.

DIASTOLIC MURMURS may be heard in the arteries in cases of extensive aortic regurgitation. In rare cases a double murmur has been heard in the femoral artery in pregnancy without aortic regurgitation. The statement has been made that it also occurs in lead poisoning and in mitral stenosis.

B. VENOUS MURMURS.—A murmur or humming sound may be heard over the great vessels above the clavicles behind the sterno-cleido-mastoid muscle in about fifty per cent. of young persons, the frequency diminishing with age. The occurrence of a murmur at this site is due to the sharp bend forward in the internal jugular vein and its relation to the omohyoid muscle (Hamernyk). This murmur is continuous, and varies in intensity. It is loudest during and immediately after auricular diastole, during inspiration and when the patient is sitting or standing, as at this time the current in the veins is strongest. It becomes very faint or disappears entirely when the patient is recumbent. It is loudest on the right side of the neck, and is increased by slightly turning the head to the left.

In anæmic conditions this physiological murmur becomes constant, intensified, and in quality more musical. It may also be induced by slight pressure over the veins in other regions of the body. The anæmic venous hum (bruit du diable) is differentiated from the arterial murmur by being continuous, and the intensity not varying with systole of the heart. When communication occurs between the artery and the vein (arterial varix), a systolic murmur is heard over the area involved.

Sounds Made in the Pericardium.—Normally, the serous surfaces covering the heart move noiselessly. When the surfaces are roughened audible vibrations are made, and the sound is recognized as pericardial by *(a)* its character, *(b)* its time, *(c)* its localization and transmission, *(d)* modifications that occur by posture and pressure.

The character of the pericardial friction sounds may simulate very closely the endocardial murmurs, but usually they are of a rubbing, scratching, rasping or scraping quality. Friction sound, although dependent upon cardiac action, may not coincide in rhythm with the different phases of the cardiac cycle, extending as a single sound beyond the period of systole or diastole. It may occur as a to-and-fro (double) sound, due to the motion of both ventricular systole and diastole, or it may even have a triple rhythm, due to auricular systole, as well as the ventricular action.

The friction sound may present variations in rhythm and quality over different portions of the præcordium, and those having a murmurish quality may lead to the diagnosis of endocardial murmur.

Pericardial friction sound is limited to the præcordial space, although rarely heard over the entire pericardium, even in general inflammation of the sac. It may be heard over that portion of the pericardium that is reflected over the base of the aorta, and be detected as high as the second interspace and as far as the left nipple. The point of maximum intensity is usually over the base of the heart, in the third and fourth interspaces.

The important diagnostic feature of pericardial sounds is that they give the impression of being made close to the surface, and that their intensity and character may be modified by posture. When the body is thrown forward so that the heart is in closer contact with the anterior portion of the pericardium, the sounds become more distinctly frictional in character, which lessens as the patient resumes the upright position, and may be absent when he is recumbent. Firm pressure with the ear or stethoscope over the præcordia also increases the intensity of the sound.

Pericardial friction sounds vary in character and location from time to time, according to the nature of the changes in the sac.

Pleuro-Pericardial Friction Sounds.—Pleuro-pericardial friction sounds may be heard over that portion of the pleura that overlaps the heart. This sound is most frequently heard in the fourth and fifth intercostal spaces. The quality and rhythm of this friction sound may correspond with that made in the pericardium, but the rhythm is disturbed by the respiratory movements, disappearing with full inspiration and recurring with expiration.

Cardio-Pulmonary Sounds.—A blowing sound, as of a murmur, may be produced by cardiac action in the layer of lung that is interposed between the heart and the chest wall. It is due to the impact of the heart forcing air out of the alveoli with sufficient force to produce a localized respiratory murmur or puff (cardio-respiratory murmur). It is heard most frequently over the apex of the heart and along the left border of relative cardiac dullness, and *is not* transmitted in the area of cardiac murmurs heard in the same locality. While caused by cardiac action, the murmur is not always synchronous with the cardiac sounds; it may be heard during systole and for a short time after the occurrence of the second sound. The intensity of the murmur is variable, and is affected chiefly by the respiratory movements, and also by the posture of the patient. It may be loudest either during inspiration or expiration, or may be present only when the lungs are fully inflated and the breath is held. At times, instead of being heard with each heart beat, it is only detected with each fourth beat, its rhythm being determined by the respiratory cycle.

Its intensity may be increased by firm pressure with the ear of stethoscope over the præcordia.

When the air-vesicles and bronchioles contain secretion, the cardiac respiratory murmur may be attended with fine crackling or moist râles.

CHAPTER XII.

DIAGNOSIS OF DISEASES OF THE HEART.

VALVULAR LESIONS.

REGURGITATION AT THE MITRAL ORIFICE.

Regurgitation at this orifice is the most frequent of all valvular lesions, and at the same time that most compatible with good physical health.

Normally, competency at the mitral valve is dependent upon *(a)* perfect coaptation of the valve leaflets; *(b)* control over the position of the valve leaflets through the chordæ tendineæ by the papillary muscle; *(c)* contraction of the muscle of the ventricle, which reduces the area of the auriculo-ventricular opening to one-half the size that it has during ventricular diastole, and also changes the shape from a circular to a somewhat oval form.

Incompetency may be due to disturbance in manner or time of one or more of these co-ordinating factors. Coaptation of the mitral leaflets may be interfered with (1) by swelling or vegetations (acute or subacute endocarditis), (2) by loss of substance (ulcerative endocarditis), (3) by thickening, shriveling or distortion, or binding together in a manner to prevent closure. (4) The fibrous ring to which the valve is attached may be so thickened as to interfere with the action of the valves. (5) The chordæ tendineæ may be stretched, thickened or distorted. (6) The papillary muscles may fail to exert the right amount of traction at the right time on the chordæ tendineæ from weakness of the muscles or change (dilatation) of the ventricular cavity. (7) The size of the auriculo-ventricular orifice may not be sufficiently contracted at the time of systole, on account of fibrous thickening of the ring at the base of the valves, flabbiness of the cardiac muscle or dilatation of the left ventricle. In this latter condition the valves, though normal, are insufficient to close the opening (relative valvular insufficiency).

Insufficiency at the mitral orifice, due to dilatation of the ventricular cavity, is a frequent secondary lesion in primary aortic stenosis or regurgitation. Imperfect ventricular contraction and dilatation of the ventricular cavity may be caused by anæmia, and by acute or chronic wasting diseases when associated with physical exertion or high blood pressure in the aorta.

Effects of Mitral Regurgitation.—The effects of mitral regurgitation are both immediate and remote, and vary in degree according to the amount of blood that is regurgitated into the auricle, the force of the current, and the extent to which the disturbance of function at the mitral orifice is compensated for. The changes induced by this lesion can be divided into three stages, according as (1) they are limited to the left auricle and ventricle; (2) as they extend beyond the left auricle and involve the pulmonary circulation and right ventricle; (3) when failure of compensation by the right ventricle occurs, with regurgitation at the tricuspid valve and interference with venous circulation.

FIRST STAGE. COMPENSATION BY LEFT AURICLE AND VENTRICLE.—The earliest effect of regurgitation is to dilate the auricle, which receives the blood from two sources: (1) Normal, the flow from the pulmonary vein; (2) abnormal, flow from the left ventricle through the mitral valve.

The increased amount of blood received into the auricle during diastole increases its work during systole, and hypertrophy occurs; and, as the dilated and hypertrophied auricle throws more blood than normally into the ventricle during its diastole, the ventricle also dilates and meets the increased demands upon it by hypertrophy. In this stage the compensation is through the dilatation and hypertrophy that occurs simultaneously in both left auricle and ventricle. If the amount of regurgitant blood is not large, there is no interference with pulmonary circulation; neither is the blood forced back into the pulmonary veins during auricular systole, as the openings of the veins are closed, as in the normal condition, by muscular contraction, and consequently the effects of regurgitation are limited to the left auricle and ventricle.

SECOND STAGE. COMPENSATION BY RIGHT VENTRICULAR HYPERTROPHY.—When the amount of regurgitant blood is large and the force of the current strong enough, dilatation of the left auricle occurs beyond the power of compensation. The auricle is quickly filled by the blood from the ventricle, and, as the pressure in the pulmonary veins is normally low, the flow of blood from the lung is interfered with, and congestion occurs. This congestion causes interference with the flow of blood through the capillaries and raises the blood pressure in the pulmonary artery, which in turn causes the pulmonary valves to close with greater tension, and induces hypertrophy of the right ventricle as its work is increased, having to empty itself against the raised pressure in the pulmonary artery. The hypertrophy of the right ventricle compensates for the lesion at the mitral orifice, and increases in degree up to a point which depends upon the nutrition of the heart muscle and the strength of the tricuspid valves.

THIRD STAGE. FAILURE OF RIGHT VENTRICULAR COMPENSATION.—When pressure in the pulmonary artery becomes raised to a certain point, compensation by the right ventricle may fail in two ways: (1) The tricuspid valve may yield to the high endocardial pressure (safety valve action), (2) or the muscular power of the right ventricle being insufficient to overcome the pressure in the pulmonary artery, the ventricle only partially empties itself during systole. At the next auricular systole it is overdistended by the blood received in addition from the right auricle. The dilatation of the right ventricular wall increases the auriculo-ventricular opening, which becomes too large to be closed by the tricuspid valves (relative insufficiency). Incompetency at the tricuspid orifice relieves the work of the right ventricle, but causes interference with venous circulation, resulting in passive congestion of the abdominal organs, and later, effusion into the serous sacs and cellular tissue.

Physical Signs. INSPECTION.—The signs noted on inspection correspond to the effects produced by the lesion upon the heart and to the secondary changes in the lungs and in the pulmonary, venous and arterial circulations. The severity of the lesion is estimated by the extent of these changes.

First Stage.—When the amount of regurgitation is slight, inspection may be negative, or dilatation and hypertrophy of the left ventricle may cause the visible apex beat to be displaced slightly downward and to the left. When mitral regurgitation occurs early in life, the apex may be carried to the left nearly as far as the anterior axillary line, and there may be bulging of the præcordia.

Second Stage.—According to the degree of right-side cardiac hypertrophy, the area of the apex beat is increased. It is seen further to the left, and also over the lower portion of the præcordia toward the median line and in the epigastrium. In marked dilatation and hypertrophy of the left auricle, pulsations may be present at the second left interspace, dependent upon the systole of the auricle. This auriculo-ventricular systole may impart a wave-like motion, from above downward, to the præcordia.

Third Stage.—When compensation, through hypertrophy of the right ventricle, fails, and dilatation results, a diffused cardiac impulse is present over the entire lower portion of the left side of the thorax, and also in the epigastrium. In extreme dilatation of the right ventricle, pulsations may be seen from the third to the sixth interspaces near the sternum on the left side, and in the epigastrium along the border of the ribs.

Tricuspid regurgitation and dilatation of the right auricle may cause pulsations to be seen in the fifth right interspace along the sternum, and systolic pulsations in the veins of the neck.

Involvement of the lungs is shown by cyanosis, dyspnœa (orthopnœa); venous stasis by enlargement of the abdomen (ascites), changed respiratory movements (hydrothorax) and œdema of the extremities.

PALPATION.—The character of the cardiac action detected by palpation corresponds with that noted by inspection.

First Stage.—When dilatation of the left ventricle is slight, the apex beat, in addition to being displaced to the left and slightly downward, is strong, and the point of contact is well defined. In proportion to the dilatation and hypertrophy, the strength of the impulse is increased and heaving in character, while the area becomes enlarged. No thrill will be felt unless mitral regurgitation is associated with stenosis.

Second Stage.—The impulse over the lower portion of the præcordia is forcible, and the area is diffused, but not so well defined.

Third Stage.—With failure of right-side compensating hypertrophy, the impulse becomes more diffused. When dilatation predominates, it has a wave-like, indistinct character, and may be detected to the right of the sternum and in the epigastrium.

The pulse in the first stage shows no alteration. In the second stage it may become very irregular. With failure of the right heart (third stage), the arteries may be improperly filled, and the pulse becomes small and irregular in force and frequency.

When incompetency of the tricuspid valve occurs, the liver will be found enlarged and extending below the free border of the ribs. In extreme cases it will be pulsating. Examination of the abdomen will show ascites, and effusion into the pleural cavities will cause enlargement of the lower portion of the thorax, with loss of respiratory movements.

PERCUSSION. *First Stage.*—Percussion shows but slight increase in the area of cardiac dullness. In hypertrophy and dilatation of the left ventricle alone, the outline of dullness over the apex is pointed.

Second Stage.—With hypertrophy of the right ventricle, the area of cardiac dullness is increased to the left at the level of the fourth rib, and the outline at the apex becomes rounded.

Third Stage.—With dilatation of the right ventricle and tricuspid regurgitation, there is increase in the upper border of flatness at the level of the fourth rib, and dullness may be detected to the right of the sternum at this point. Over the lower portion of the sternum there may be diminution of the sternal resonance; at the apex dullness extends to the left and downward.

Pulmonary Resonance.—In the first and second stages the pulmonary resonance is not impaired. In the third stage, œdema of the base of the lungs may cause a diminished resonance. In the later stages, with effusion into the pleural cavity (hydrothorax), flatness is present over the lower portion of the chest. The percussion area of the liver is also increased.

AUSCULTATION.—The distinctive sign of regurgitation at the mitral orifice is a murmur which occurs with the ventricular systole, accompanies, replaces or follows the first sound of the heart (Figs. 50 and 51), having its point of maximum intensity at or near the apex, and is diffused in all directions, especially to the left, to the axillary line or beyond. Posteriorly, it may be heard in the left interscapular space, just below spine of scapula.

The quality of the murmur is variable. It may be harsh, high pitched and whistling in character, or soft, low pitched and blowing. Some claim that it always has a more or less musical quality. It also changes from time to time, and is influenced by posture, being most marked when the patient is in the recumbent position.

The quality and intensity of the murmur do not indicate the extent nor character of the lesion, which are estimated by the effect upon the heart and changes in the normal cardiac sounds, and in the circulatory system.

First Stage.—In slight regurgitation a murmur may not be present, but the first sound of the heart at the apex may be prolonged, due to change in the valvular element (impure first sound). When a murmur accompanies the first sound which retains its valvular element, the amount of regurgitation is small. The distinguishing sign of this stage is the absence of accentuation of the second sound of the heart in the pulmonic area, with a normal intensity of the aortic sound; also the absence of right-side ventricular hypertrophy.

Second Stage.—On the other hand, accentuation of the pulmonic second sound is a characteristic sign of compensation of the mitral lesion by hypertrophy of the right ventricle. It has been claimed that the diagnosis of mitral regurgitation cannot be made unless this accentuation of the pulmonic second sound is present.

In estimating the extent of the lesion by the accentuation of the pulmonic second sound and its relation to the aortic, the age of the patient should always be taken into consideration; also whether increased tension in the pulmonary artery is due to obstructive pulmonary disease (see page 274).

When right-side ventricular hypertrophy compensates for the lesion at the mitral valve, the murmur retains a fairly definite quality, intensity and duration; also auscultation of the lungs does not show evidences of pulmonary œdema, although the breath sounds may be harsher than normal.

Third Stage.—With failure of compensation, the murmur at the apex becomes less intense. Its transmission to the left is less marked, while the occurrence of tricuspid regurgitation causes the murmur to be heard over a wider area to the right, it often being impossible to separate these two murmurs; at the same time the pulmonic second sound diminishes in intensity. The mitral murmur in some cases may entirely disappear, the tricuspid murmur only being heard. In proportion to the failure of compensation, the physical signs of pulmonary œdema and effusion into the pleural cavities occur.

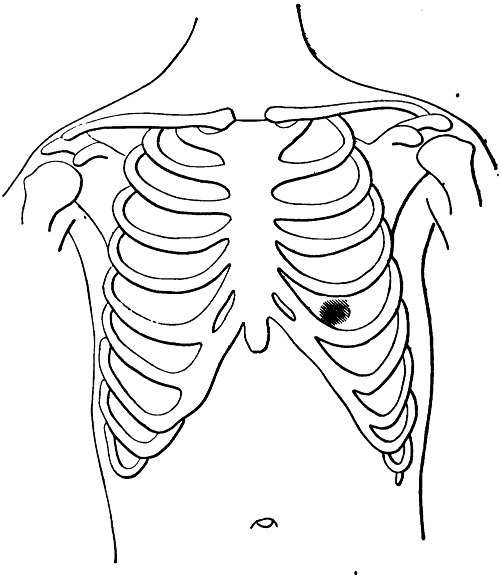
Diagnosis of the Pathological Condition.—The nature of the lesion at the mitral orifice cannot be determined by the physical signs alone, although these may be in a measure distinctive. When incompetency is due to a *recent* endocarditis, there is very little change in the size of the heart, and the murmur is of a soft, blowing quality. When the valves are distorted, thickened, or perfect coäptation prevented by contraction of the chordæ tendineæ, due to a previous endocarditis, the effect upon the left and right heart is more marked, and the murmur has a harsh, rough and more musical quality.

According to the degree of interference with the valves, the murmur replaces the valvular element of the first sound at the apex.

Incompetency due to dilatation of the left ventricle (relative valvular insufficiency) rarely gives a loud, high-pitched murmur. When this condition is secondary to disease of the aortic orifice, or changes in the arterial system, the mitral murmur is much less intense than those at the aortic orifice. Age is an important factor in determining the nature of the lesion. Under forty years of age, uncomplicated mitral regurgitation is generally due to a previous endocarditis. Occurring after forty-five years of age, it is generally due to secondary dilatation, and is associated with the history and physical signs of the primary lesion at aortic orifice or in arteries (arterio-sclerosis).

Functional and Hæmic Murmurs in the Mitral Area.—Murmurs with the first sound at the apex, that occur in anæmia or severe febrile diseases, are generally soft, blowing in quality, and are not associated with secondary changes in the heart or pulmonary circulation. (Fig. 62.) The apex beat is not displaced; the force is weak, not lifting; the second sound over the pulmonic area is not accentuated, although there may be a soft, blowing murmur (Fig. 63), and the tension of the radial pulse is unaffected.

FIG. 62.

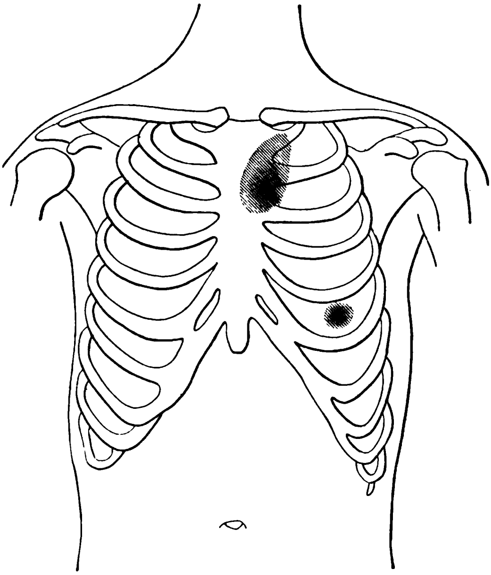


Anæmia—systolic murmur in mitral area.

Murmurs of this character may occur during acute febrile diseases, especially rheumatism, and be due to acute endocarditis affecting the mitral valve, or to temporary insufficiency resulting from cardiac muscular weakness (myocarditis). It is impossible to determine whether these murmurs are organic or functional at first examination, and their nature can only be decided by noting the changes that occur in the murmur and the effect on the heart and circulation.

Aortic stenosis may be attended by a murmur that is heard at the apex, with a quality different from that present at the base of the heart, and simulating the murmur of mitral regurgitation. This murmur is not transmitted to the axilla nor associated with accentuation of the pulmonic second sound.

FIG. 63.



Anæmia, systolic murmur in mitral and pulmonary areas.

Differential Diagnosis. — Exo-cardial murmurs may simulate those of mitral regurgitation. *Pericardial* *friction sound* may be localized at the apex, and be synchronous with the first sound of the heart. It is differentiated from the endocardial murmur by changes in the intensity and character caused by pressure and position, and it is not transmitted beyond the apex beat nor heard behind in the interscapular space.

Pleuritic friction sounds generally have more or less of the friction quality, and are influenced also by pressure and position, and their rhythm is more dependent upon respiratory than cardiac action.

Cardio-respiratory murmurs are also influenced by the respiratory movements, being intensified during inspiration, and generally inaudible at the end of expiration. The point of maximum intensity does not correspond to the apex of the ventricle, but is somewhat removed from it. The murmur does not replace the first sound of the heart nor change its valvular character. It is also influenced by the position of the patient, being most marked in the recumbent position, although this is not an invariable rule.

STENOSIS AT THE MITRAL ORIFICE.

Obstruction at the mitral orifice may occur as an isolated lesion, but it is usually associated with regurgitation.

Normally, with ventricular diastole the mitral orifice enlarges to twice the size that it has during systole, and the relaxation of the papillary muscles allows free opening of the leaflets. The flow of blood through the opening is due to three forces that are operative at different periods of the ventricular diastole: (1) The aspirating or suction force of the dilating ventricle. This force is most marked during the first period of ventricular diastole, and gradually decreases as the ventricle becomes distended. (2) Pressure of blood in the auricle and pulmonary veins. This is most marked also at the beginning of diastole. (3) Auricular systole, which completes the emptying of the auricle, and occurs just before the first sound of the heart.

Narrowing of the mitral orifice may be due to changes that involve the auriculo-ventricular ring, interfering with the normal enlargement of the opening during diastole, or they may involve the leaflets or the chordæ tendineæ. These changes are generally secondary to endocarditis.

The earliest change of endocarditis is usually the production of vegetations on the auricular surface of the valve, which, while causing no obstruction, render the surface rough and allow of the production of a murmur. Secondary thickening of the leaflets and the chordæ tendineæ prevent free motion of the valves, causing them to project into the centre of the ventricle, and, while the obstruction is slight, fluid veins are formed. Attachment of the edges of the leaflets may occur, converting the valve into a funnel, and producing more or less interference with the flow of blood. This condition is especially liable to occur in children. More marked adhesion and retraction of the valves may occur, so that the opening becomes a mere split.

Effects of Mitral Obstruction.—The changes that occur as the result of mitral stenosis are comparatively slow, and the effects produced upon the circulatory system can be divided into three stages.

First Stage.—The immediate effect of obstruction at the mitral orifice is to increase the work of the left auricle, which is met by a compensating hypertrophy.

When the obstruction to the flow of blood is slight, the effect may not extend beyond a simple hypertrophy of the left auricle, which is sufficient to force the normal amount of blood into the ventricle and to prevent any interference with the pulmonary circulation.

Second Stage.—When the obstruction is more marked, the effect may extend beyond the left auricle and involve the pulmonary circulation and the right heart. Interference with the pulmonary circulation may occur in two ways, according as the hypertrophy of the left auricle is sufficient or insufficient to compensate for the obstruction. (1) Hypertrophy of the left auricle may be sufficient to close the openings of the pulmonary veins and to empty itself at the time of auricular systole, and yet pressure in the pulmonary circulation may be above normal. This occurs when the obstruction at the mitral orifice is so marked that the aspirating force of the heart and the pressure in the pulmonary veins are insufficient to empty the pulmonary circulation during the early portion of diastole. This increases pressure in the pulmonary veins, arteries and capillaries, causing secondary hypertrophy of the right ventricle, with accentuation of the pulmonic second sound. Dilatation of the left auricle is not present during this period of the second stage, and only occurs when the power becomes insufficient to overcome obstruction.

(2) When the hypertrophy of the left auricle is unable to overcome the obstruction, dilatation occurs, and with auricular systole the blood is forced from the auricle, not only through the obstruction, but also into the pulmonary veins, and marked congestion of the lung ensues. As was explained under "Mitral Regurgitation," this interference with pulmonary circulation is compensated for by hypertrophy of the right heart.

During this stage the left ventricle may be normal, or, on account of imperfect distension of its cavity during diastole, this cavity may be reduced in size.

Third Stage.—Failure of right ventricular compensation occurs in mitral obstruction under the same conditions and with the same effects as has already been noted under "Mitral Regurgitation."

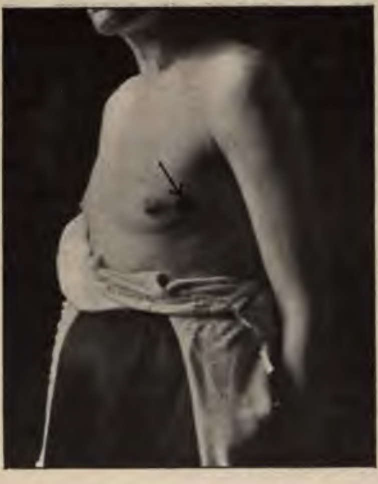
Physical Signs. INSPECTION.—The changes noted by inspection correspond to change in the position of the apex beat and the condition of the circulation.

In the *first stage,* there being no increase in the size of the ventricles, the apex beat is in the normal position. In children the pulsation of the auricle may be noted in the second and third left interspaces.

In the *second stage*, especially during the early period, the apex beat is displaced upward and to the left, even beyond the nipple line. (Fig. 64.) With dilatation of the right ventricle there is increase in the area of visible apex beat and extension toward the median line. When the disease occurs in early life, there may be slight enlargement of the præcordia.

In the *third stage* the apex beat is diffused and indistinct. Interference with the pulmonary circulation causes marked cyanosis of the membranes and a dusky appearance of the skin. Interference with return circulation causes the veins of the neck to become prominent, and pulsations are noted when tricuspid regurgitation is present.

FIG. 64.



Displacement of apex upward and to the left in mitral stenosis.

PALPATION. *First Stage.*—The apex beat may be normal or slightly decreased in force. Over the apex beat a thrill may be felt. The characteristic feature of the thrill is its sudden termination at the moment that the apex beat is felt. The duration of the thrill and its length will depend upon the degree of obstruction and the force of the current during different periods of ventricular diastole.

The pulse may be normal or show slight emptiness of the artery.

Second Stage.—Hypertrophy of the right ventricle causes the apex beat to be more diffused. Over the left intercostal spaces the closure of the pulmonary valves may be felt as a shock. The thrill is more marked. The pulse may be normal and regular when patient is quiet, becoming irregular on exertion.

Third Stage.—With failure of right-side compensation, the apex beat becomes more diffused and wave-like, the thrill weaker or absent, or, if present, is felt but for a short time before the apex beat. The pulse is small, irregular, and the arteries are imperfectly filled.

When failure of compensation has occurred gradually, with long-continued interference with the venous circulation, increased pressure in the arteries, due to obstruction to capillary circulation, may cause hypertrophy of the left ventricle, so that the apex beat will be more marked than during the earlier stages.

PERCUSSION. *First Stage.*—The area of percussion dullness is normal. During the second and third stages it corresponds to the anatomical enlargement of the heart (see Mitral Regurgitation).

AUSCULTATION.—The diagnostic sign of obstruction at the mitral orifice is the presence of a marked vibratory murmur, which begins an appreciable time after the second sound and increases in intensity until terminated abruptly by the accentuated first sound (presystolic); the point of maximum intensity is at the apex, around which it is diffused.

The above are characteristics of the typical murmur of mitral obstruction, but it is subject to variations in quality, duration and intensity, dependent upon the degree and character of the obstruction and the force of the current of blood through the opening.

The murmur has been described as rough, rumbling, rolling, churning, grinding, blubbering, and may be simulated by pronouncing the syllables "rrb," "rrt."

The most distinctive feature is the short, sharp sound that terminates the murmur, which always persists, even though the other phases of the murmur are absent. It may be the only sign present of mitral obstruction, as the murmur is not constant.

The production of a murmur demands that the blood pass through the contracted orifice with a definite amount of force. As the rapidity of the passage of the blood through the heart varies from time to time, according to the rate of cardiac action, the murmur may be absent at one time and present at another. Variations in the force of the current also cause a rise and fall in the intensity of the murmur.

The duration of the murmur and its relation to the first and second sounds of the heart also vary. When the murmur is heard only for a brief period, just before the first sound, it is distinctly pre-systolic. The duration may be longer, however, so that it occupies the major portion of diastole, or in some cases it may be heard with greatest intensity almost immediately after the occurrence of the second sound; but this murmur can never occur with or replace the second sound of the heart. (Fig. 47.)

The presence of a murmur during the different portions of diastole depends upon the nature of the force. When the force necessary to produce the murmur is due to auricular systole, the murmur is heard just before the first sound. When the constriction is marked and the force is supplied by the aspirating action of the left ventricle and the tension that exists at the time in the auricle and pulmonary veins, the murmur will occur earlier in diastole.

First Stage.—The murmur is harsh in proportion to the thrill is its sudden termination at the moment that the apex beat is felt. The duration of the thrill and its length will depend upon the degree of obstruction and the force of the current during different periods of ventricular diastole.

The pulse may be normal or show slight emptiness of the artery.

Second Stage.—Hypertrophy of the right ventricle causes the apex beat to be more diffused. Over the left intercostal spaces the closure of the pulmonary valves may be felt as a shock. The thrill is more marked. The pulse may be normal and regular when patient is quiet, becoming irregular on exertion.

Third Stage.—With failure of right-side compensation, the apex beat becomes more diffused and wave-like, the thrill weaker or absent, or, if present, is felt but for a short time before the apex beat. The pulse is small, irregular, and the arteries are imperfectly filled.

When failure of compensation has occurred gradually, with long-continued interference with the venous circulation, increased pressure in the arteries, due to obstruction to capillary circulation, may cause hypertrophy of the left ventricle, so that the apex beat will be more marked than during the earlier stages.

PERCUSSION. *First Stage.*—The area of percussion dullness is normal. During the second and third stages it corresponds to the anatomical enlargement of the heart (see Mitral Regurgitation).

AUSCULTATION.—The diagnostic sign of obstruction at the mitral orifice is the presence of a marked vibratory murmur, which begins an appreciable time after the second sound and increases in intensity until terminated abruptly by the accentuated first sound (presystolic); the point of maximum intensity is at the apex, around which it is diffused.

The above are characteristics of the typical murmur of mitral obstruction, but it is subject to variations in quality, duration and intensity, dependent upon the degree and character of the obstruction and the force of the current of blood through the opening.

The murmur has been described as rough, rumbling, rolling, churning, grinding, blubbering, and may be simulated by pronouncing the syllables "rrb," "rrt."

The most distinctive feature is the short, sharp sound that terminates the murmur, which always persists, even though the other phases of the murmur are absent. It may be the only sign present of mitral obstruction, as the murmur is not constant.

The production of a murmur demands that the blood pass through the contracted orifice with a definite amount of force. As the rapidity of the passage of the blood through the heart varies from time to time, according to the rate of cardiac action, the murmur may be absent at one time and present at another. Variations in the force of the current also cause a rise and fall in the intensity of the murmur.

The duration of the murmur and its relation to the first and second sounds of the heart also vary. When the murmur is heard only for a brief period, just before the first sound, it is distinctly pre-systolic. The duration may be longer, however, so that it occupies the major portion of diastole, or in some cases it may be heard with greatest intensity almost immediately after the occurrence of the second sound; but this murmur can never occur with or replace the second sound of the heart. (Fig. 47.)

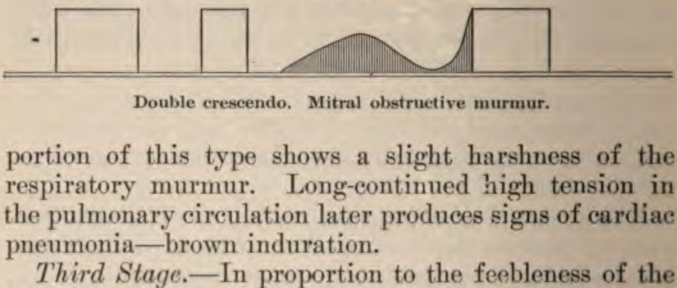
The presence of a murmur during the different portions of diastole depends upon the nature of the force. When the force necessary to produce the murmur is due to auricular systole, the murmur is heard just before the first sound. When the constriction is marked and the force is supplied by the aspirating action of the left ventricle and the tension that exists at the time in the auricle and pulmonary veins, the murmur will occur earlier in diastole.

First Stage.—The murmur is harsh in proportion to the constriction and the auricular hypertrophy, markedly pre-systolic, and terminated with the accentuated first sound of the heart, which is clear. In slight stenosis the accentuated first sound may only be present, or the murmur may be temporarily absent when the patient is in bed and the cardiac action is quiet.

Second Stage.—The murmur is harsh, occurs earlier in the diastole, with marked pre-systolic increase in intensity. The pulmonic second sound is accentuated, and there is a tendency to reduplication of the second sound over the base of the heart and absence of the second sound at the apex. Change in the second sound at the base or apex is due to increased pressure in the pulmonary artery, with diminished tension in the aorta.

During this stage, when the power of the left auricle is failing, two points of intensity may be noted (double crescendo); one mid-diastolic, and the other pre-systolic, depending upon the varying force of the blood current. (Fig. 65.) Auscultation of the lung during the early portion of this type shows a slight harshness of the respiratory murmur. Long-continued high tension in the pulmonary circulation later produces signs of cardiac pneumonia—brown induration.

FIG. 65.



Double crescendo. Mitral obstructive murmur.

Third Stage.—In proportion to the feebleness of the left auricular systole and lowering of the pressure in the pulmonary circulation, due to failure of right heart compensation, the murmur loses its characteristic roughness or disappears. Accentuation of the second sound is absent. The first sound becomes short, sharp and sudden over the tricuspid area; the soft systolic murmur of tricuspid regurgitation may be detected. Over the lungs, failure of the right heart is shown by the physical signs of pulmonary œdema and effusion into the serous cavities, as noted under "Mitral Regurgitation."

Differential Diagnosis.—Murmurs that may be mistaken for that of mitral stenosis are (1) those whose points of maximum intensity are at the apex—mitral regurgitation, tricuspid regurgitation—and (2) those that occur during diastole—aortic regurgitation (Flint murmur), tricuspid stenosis, pericardial (friction) murmurs.

MITRAL REGURGITATION.—The murmur of mitral stenosis increases in intensity up to the occurrence of the first sound of the heart; that of mitral regurgitation begins with the first sound, and gradually diminishes in intensity.

The murmur of mitral stenosis is localized at the site of the visible apex beat; that of mitral regurgitation is transmitted beyond the præcordia toward the axilla, and is heard also behind.

Mitral regurgitation and mitral stenosis are frequently associated. Under these circumstances, when both murmurs are present, a harsh, rough murmur is heard, culminating with the sharp first sound, which is immediately followed by a softer, blowing murmur, which is transmitted toward the axilla.

TRICUSPID REGURGITATION.—The murmur of tricuspid regurgitation may be heard with the point of maximum intensity just within the apex beat. When not secondary to mitral stenosis, its time, its area of diffusion and the secondary effects upon the venous circulation are the distinctive features. When associated with mitral stenosis, it follows the first sound of the heart, and is soft, blowing in character. Tricuspid regurgitation is determined more by the changes in the venous circulation than by the presence of the murmur.

AORTIC REGURGITATION.—The murmur of mitral stenosis may occupy most of the diastolic period. It usually begins an appreciable time after the occurrence of the second sound, and can never accompany or replace it. The murmur of aortic regurgitation, on the other hand, accompanies, replaces or follows immediately after the aortic sounds.

The murmur of mitral stenosis is suddenly terminated with the occurrence of the first sound. That of aortic regurgitation is most marked at the beginning of diastole, and usually fades out before the occurrence of the first sound.

"FLINT" MURMUR.—This may occur as a marked presystolic increase of the diastolic murmur, or may occur as a separate sound in aortic regurgitation.

TRICUSPID STENOSIS.—The time of these two murmurs are identical. Their points of maximum intensity may be slightly separated, but usually they are the same. The murmurs are also similar in quality and duration during the diastolic period. The differentiation is extremely difficult, and is made only when the two murmurs vary in point of maximum intensity and in quality.

PERICARDIAL FRICTION SOUNDS.—Pericardial friction sounds, especially when due to adherent pericardium, may simulate the murmur of mitral stenosis, but the sound does not terminate with the apex beat, being carried slightly into the systolic period.

OBSTRUCTION AT THE AORTIC ORIFICE.

AORTIC STENOSIS.

Obstruction to the flow of blood through the aortic orifice, due to diminution of its calibre, is a rare condition, but signs somewhat similar to those produced by narrowing of the orifice are present when the orifice remains of the same diameter as in health.

True obstruction, or stenosis, at the aortic orifice may be caused by (1) contraction of the fibrous ring; (2) rigidity of the cusps of the valves; (3) adhesions of the borders of the valves, and (4) masses of vegetations.

Non-obstructive conditions producing a murmur at the aortic orifice may be (1) roughening of the surface of the valves by vegetations, calcareous deposits; (2) changes in the size of the aorta (relative obstruction), and (3) changes in the specific gravity of the blood (hæmic or functional murmur).

Effect of Stenosis.—The effects of obstruction at the aortic orifice may be divided into three stages, according as they are limited to (1) the left ventricle, (2) extend beyond the mitral valve and involve the pulmonary circulation and right ventricle, (3) when they extend beyond the tricuspid valve and involve the venous circulation.

First Stage.—As the lesions that produce obstruction, whether due to endocarditis or secondary changes in the aorta, progress slowly, there is a corresponding gradual increase in the work of the left ventricle, which is mėt by compensating hypertrophy without any corresponding degree of dilatation. Dilatation does not occur until the left ventricle fails to empty its cavity at each systole. This failure may be temporary, as when the cardiac rate is suddenly increased. Compensation by increased hypertrophy may again occur, but the dilatation of the cavity remains.

The effect of the stenosis is limited to the left ventricle for a variable period until repeated, or extensive dilatation of the left ventricular cavity causes imperfect action of the papillary muscles and enlargement also of the auriculo-ventricular ring and relative insufficiency of the mitral valves. The effect upon the arterial system is marked, producing a characteristic pulse.

Second Stage.—When regurgitation at the mitral orifice occurs the work of the left ventricle is temporarily relieved, and there is immediate interference with the pulmonary circulation, increased tension in the pulmonary artery and compensating hypertrophy of the right ventricle. For subsequent changes in this stage, and those that occur during the third stage, with failure of compensation by the right ventricle (see Mitral Regurgitation, page 304).

In conditions which give a similar murmur, but are non-obstructive, the primary effect on the left ventricle and the subsequent changes in the heart are absent. An exception to this is where atheroma or loss of elasticity of the aorta has preceded the dilatation of the aorta, when there is hypertrophy of the left ventricle. In this condition dilatation of the aorta may occur, instead of regurgitation, at the mitral orifice.

Physical Signs. INSPECTION.—The position and extent of the apex beat correspond to the changes in the size of the heart.

First Stage.—When there is hypertrophy only of the left ventricle, the apex beat may be in the normal position, or, at most, carried slightly to the left, and is well defined. When hypertrophy and dilatation are associated, it is displaced to the left and downward, and the area is more diffused.

Second Stage.—The apex beat is seen further to the left, and also over the lower portion of the præcordia. Visible pulsations may be present in the third, fourth and fifth interspaces (left), and there may be præcordial bulging.

Third Stage.—Dilatation of the right ventricle, venous engorgement, and tricuspid regurgitation give a diffused, wavy pulsation, with enlargement and pulsations of the veins in the neck.

PALPATION. *First Stage.*—Palpation gives important information concerning not only the condition of the heart, but also the degree of stenosis and the nature of the structural changes. The apex beat is characteristic. It lacks the sudden, sharp tap of the normal heart, and the impulse is slow, labored and sustained, terminating with a short, sharp recoil that may give the sensation of a slight tap.

When dilatation is added to hypertrophy, the labored and forcing action of the heart is increased, but it does not have the lifting, heaving impulse of hypertrophy and dilatation due to other causes. Over the base of the heart an intense thrill may be felt; the point of maximum intensity is usually in the aortic area, but it may be detected also at the apex.

The intensity of the thrill does not indicate the degree or nature of the lesion, although it occurs most frequently when the obstruction is due to fibrous or atheromatous thickening of the aortic ring or cusps. The intensity of the thrill is more directly connected with the force of the current.

The pulse is of great importance in aortic stenosis. Its character, when taken in connection with the changes in the heart, determines the extent of the lesions that cause the systolic murmur at the aortic orifice.

In proportion to the degree of obstruction is the variance between the slow, labored, but forcible, apex beat and the small pulse wave.

Normally, with ventricular systole the blood is rapidly forced into the aorta, which is distended, giving to the radial artery the characteristic pulse wave. In proportion to the obstruction the blood is slowly forced into the aorta during the entire period of systole, so that there does not occur the sudden distension of the aorta, and the pulse wave lacks the sharp rise and continues longer.

Non-obstructive conditions, producing aortic systolic murmurs, do not give this type of pulse.

The characteristics of the pulse in aortic stenosis are well seen in the sphygmographic tracings (Fig. 41).

The effect of exertion on the pulse is important in determining the power of the hypertrophied left ventricle to compensate for the lesion. When exertion increases the frequency and force of the pulse, then compensation is perfect, be the obstruction small or great. If it causes a long, even and regular pulse while the patient is quiet, to become shorter, small and irregular in force and rhythm, then the compensation is sufficient when the heart is beating slowly, but is inadequate to empty the ventricle when the rate is increased.

Second Stage.—When regurgitation at the mitral orifice occurs, the cardiac impulse is less labored, but shorter and more diffused. The thrill over the base of the heart becomes less intense or may disappear, and the pulse becomes shorter and smaller and is not disturbed to the same extent by exertion.

Third Stage.—The impulse becomes indistinct and wavy, the thrill is absent and the pulse is small and thready.

PERCUSSION.—Changes in the areas of cardiac flatness and relative dullness correspond to the changes in the size of the right and left ventricles. During the stage of simple hypertrophy of the left ventricle, no change in the percussion can be detected. With the occurrence of dilatation, the area of deep dullness is enlarged to the left and downward.

AUSCULTATION.—The characteristic sign of aortic stenosis is a systolic murmur, occurring with the first sound of the heart at the base,[1] with the point of maximum intensity in the second right intercostal space and transmitted upward and laterally. A marked feature of systolic murmurs made at the aortic orifice is their transmission into the great arteries. (Fig. 53.) The murmur varies in quality, duration, point of maximum intensity and area of diffusion, according to the character and extent of the lesion and the secondary changes in the heart.

First Stage.—When the obstruction is slight and due to vegetations or recent endocarditis, the murmur is soft, and occupies but a portion of the systolic period. The point of maximum intensity is at the second right intercostal space, and the area of diffusion is but slightly beyond it. The aortic second sound is only slightly diminished.

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[1] If the murmur is timed by the apex beat or the first sound at the apex, it may seem to occur slightly after the beginning of systole.

When fibrous or atheromatous changes have stiffened the valves and the aortic ring, the murmur replaces the first sound at the base, and is harsh and sawing in character, especially if thrill be present. It may persist during the entire systolic period. The second sound is muffled or impure; the point of maximum intensity is at or above the second right costal cartilage, and the murmur is heard over the upper portion of the sternum, laterally along the ribs and clavicles, and may be heard in the interscapular space behind.

When dilatation of the left ventricle occurs, the point of maximum intensity is at the left edge of the sternum, at the junction of the third and fourth costal cartilages. A systolic murmur may also be heard at the apex, which may be due to conduction of the murmur made at the base through the ventricular wall, but usually caused by slight regurgitation at the mitral orifice. When due to mitral regurgitation, at first the murmur may be only present after exertion or other causes that increase the cardiac rate.

Second Stage.—The murmur at the base loses its harshness. Its area of diffusion is less, and the murmur at the apex becomes more marked; it is associated with accentuation of the pulmonic second sound and with other signs of pulmonic congestion, and later with those of the *third stage*—venous congestion and tricuspid regurgitation (see Mitral Regurgitation).

Diagnosis of the Pathological Condition.—The diagnosis of aortic stenosis from other conditions, causing a systolic murmur over the aortic area, is made by the association with the murmur *(a)* the signs of primary cardiac hypertrophy of the left ventricle, *(b)* the presence of a thrill, *(c)* the characteristic contrast between the force of the apex beat and the size of the pulse.

Aortic obstruction rarely continues as an isolated lesion for any length of time, being usually associated with regurgitation. In uncomplicated stenosis the aortic second sound is impure, muffled and lacks the normal tense, valvular element. When regurgitation occurs, the aortic sound becomes very feeble, or is replaced by the murmur.

Roughening of the surface of the valves gives a soft systolic murmur, which is localized around a point of maximum intensity at the second left intercostal space; the aortic second sound is clear, and there is no change in the position and force of the apex beat or in the pulse. These sounds are identical with the early stage of acute endocarditis at the aortic valve, that may ultimately produce true obstruction.

Dilatation of the aorta, altering the correlation between the size of the aortic orifice and the cavity beyond, frequently causes a murmur. The early changes due to atheroma diminish the elasticity of the aorta, converting it into a rigid tube, increasing the work of the heart, and causing hypertrophy of the left ventricle. If the aortic surfaces are also roughened, a harsh murmur will be present, the aortic second sound will be accentuated, and there will be more marked distension and pulsation of the vessels of the neck as a result of the loss of distensibility in the aorta. The pulse wave will be more sudden, and correspond in size and force to the apex beat. When dilatation occurs, the murmur becomes softer than that of simple aortic stenosis, the second sound is accentuated, the apex beat has the prolonged, labored character, and the pulse is sudden and short. In dilatation and relaxation of the aorta, due to neuro-cardiac conditions (Grave's disease, hysteria, etc.), the murmur is soft, blowing in quality, and heard over the vessels of the neck; the cardiac action is rapid; the first sound of the heart is short and sharp; the second sound is clear; the pulse full and compressible, and there are pulsations in the vessels of the neck.

Differential Diagnosis. ANEURISM.—Aneurism of the ascending aorta may give a systolic murmur and thrill similar to those in aortic stenosis, but the second sound is increased in intensity, and may be felt as diastolic valve shock. There is diminution of the resonance over the upper portion of the sternum, and there is generally present pulsation over the upper portion of the sternum, and pressure symptoms involving the bronchi and nerves. The pulse does not show the characteristics of aortic stenosis.

HÆMIC MURMURS having their point of maximum intensity at the second right intercostal space are heard also in the second left intercostal space, the usual site of maximum intensity. They are soft, blowing murmurs, and are not associated with signs of cardiac hypertrophy. The pulse is short, soft and compressible. The skin and mucous membranes have the characteristic appearance of anæmia.

PULMONARY OBSTRUCTION has a systolic murmur, with its point of maximum intensity to the left of the sternum, but the murmur is not transmitted into the vessels of the neck. The aortic second sound and pulse are unchanged.

PATENT DUCTUS ARTERIOSUS gives a systolic murmur, with the point of maximum intensity in the second left intercostal space. The murmur begins somewhat after the first sound (late systolic), and continues usually after the second sound. It may be heard over the entire upper portion of the chest, but is "never carried into the vessels of the neck or arms."

REGURGITATION AT THE AORTIC ORIFICE.

Regurgitation at the aortic orifice is always dependent upon structural changes in the valves guarding it, or on dilatation of the fibrous ring. Perfect closure at this orifice is dependent upon normal elasticity of the semilunar valves, whose free edges are forced together by the pressure of the blood in the aorta at the time of diastole.

Regurgitation may be caused by (1) vegetations on the free surface of the valves, preventing perfect coaptation; (2) loss of tissue due to ulcerative endocarditis; (3) the cusps may be thickened, shrunken or deformed by sclerotic processes, due to chronic endocarditis or secondary to changes in the aorta.

The sclerotic processes that produce thickening and distortion of the cusps, or changes in the aorta, are slow, but progressive, so that there is a tendency for the insufficiency of the valve to gradually increase.

(4) Dilatation of the aorta may be so marked as to involve the fibrous ring and produce enlargement of the aortic orifice, rendering it too wide to be closed by the cusps[1] (relative valvular insufficiency).

(5) Rupture of the aortic cusps may be caused by violent muscular exercise. It is doubtful whether rupture of a cusp can occur from this cause unless it has been previously diseased.

Effects of Regurgitation at the Aortic Orifice.—The effects of regurgitation at the aortic orifice can be divided into three stages, according as they are limited (1) to the left ventricle and arterial system; (2) as they extend beyond the mitral valve and involve the pulmonary circulation and right ventricle, and (3) as they extend beyond the tricuspid valve and cause interference with the general venous system.

First Stage.—The immediate effect is exerted *during diastole* on the left ventricle and arterial circulation. On the ventricle the effect of the regurgitant stream is to produce dilatation. The dilating force is due *(a)* to the pressure of the regurgitant current from the aorta, and *(b)* to that in the auricle and pulmonary veins, reinforced by auricular systole. The blood is driven into the ventricle by the elastic recoil of the aorta, and is aspirated also into the ventricle during the early portion of diastole by the expanding cavity. When the ventricle is filled, the pressure in the aorta continues to act on the left ventricle through the incompetent valve, according to Pascal's law, and cause still further dilatation. The rapid filling of the ventricle from the aorta removes one of the normal forces that aids the flow of blood from the auricle (see Mitral Obstruction, page 312), so that with auricular systole a larger amount of blood is forced into the already overfilled ventricle.

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[1] Dilatation of the fibrous ring sufficient to cause incompetency of the valve has been denied, but its occurrence must be admitted.

On the aorta an equally marked effect is produced during diastole, according to the extent of the regurgitation. There is a sudden diminution of the blood pressure, which is felt throughout the entire arterial system.

The primary effects produced during diastole are followed by secondary changes in the ventricle and orta during systole. The increased amount of blood in the ventricle augments the work of the heart during systole, which is met by a compensating hypertrophy. The greater capacity of the ventricle causes a larger amount of blood than normal to be driven into the aorta, and the hypertrophied muscle propels it with greater force. These two factors cause increase in the normal blood pressure and overdistension of the aorta and great vessels during systole, which is followed by almost as sudden a fall of blood pressure during diastole, due to aspiration into the ventricle.

The effect of the sudden forcing of an increased amount of blood into the arteries causes them to become not only dilated, but also elongated and tortuous. This effect upon the arteries may extend to the veins and capillaries.

The dilatation of the ventricle during diastole, and the hypertrophy of the ventricle and dilatation of the aorta during systole must be considered as correlated effects of aortic regurgitation. In uncomplicated regurgitation they bear a definite relation to each other. As the dilating force is constantly acting on the ventricle, there may be frequent attacks of dilatation, followed by recompensation, before insufficiency at mitral valve occurs. Under these circumstances the left ventricle may attain enormous proportions (cor bovinum).

When obstruction is associated with regurgitation, the sequence of effects is disturbed. Hypertrophy of the left ventricle is most marked, while dilatation of the aorta is less, due to the slowness with which the enlarged ventricle empties itself through the constricted opening.

Second Stage.—When the mitral valve becomes incompetent through dilatation of the ventricle or changes in the cusps of the valves, there is marked interference with pulmonary circulation and the functions of the right heart, causing the consecutive changes, as described in Mitral Regurgitation (page 303).

Incompetency of the mitral valve secondary to aortic regurgitation must not be considered as an unfavorable complication. Generally it is a conservative process, relieving the stress on the right ventricle. When compensating hypertrophy does not keep pace with dilatation through imperfect nutrition, or is ruptured by intercurrent causes, as physical exertion, disease, etc., the constantly acting dilating force tends to overpower the contracting power of the ventricle, and death from asystole occurs unless regurgitation through the mitral orifice takes place, relieving the left ventricle both during diastole and systole.

Compensation for the aortic regurgitation by hypertrophy of the right ventricle may exist for a long time. When it fails *(third stage)*, it is due to the same cause that induces it in lesions at the mitral orifice.

Physical Signs. INSPECTION.—Inspection of the præcordia and arteries gives important information, and is frequently sufficient for diagnosis.

First Stage.—When the regurgitation has produced but a small amount of dilatation and hypertrophy, the apex beat is but slightly displaced, and is localized. As the size and power of the heart increase, the apex is carried further downward and outward, and the visible impulse becomes more marked, with a diffused heaving of the chest and bulging of the præcordia. In some cases the diminution of the size of the enlarged heart during systole and the increased negative pressure are sufficient to cause retraction of the chest wall over the region of the apex beat, instead of the usual lifting impulse.

Pulsations of the aorta, carotids, subclavians, brachials and more peripheral arteries are present in proportion to the extent of the lesion. The blood-vessels also stand out more prominently, and when they are markedly tortuous the pulsations in the more superficial ones have a peculiar vermicular appearance. Pulsation in the vessels may be strong enough to produce movement of the extremities, or even of the head. In well-marked cases capillary pulsation may be present.

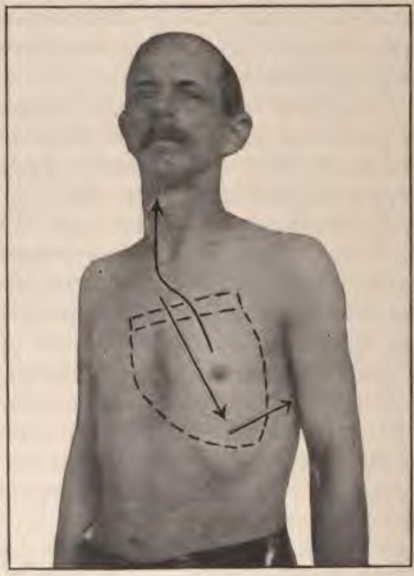
Second Stage.—The apex beat becomes more diffused, and is carried further to the left and downward. The pulsation in the vessels of the neck are not as marked.

Third Stage.—The heaving left impulse is replaced by a diffuse apex beat, extending to the epigastrium. Systolic pulsations are seen in the jugulars.

PALPATION.—Palpation of the præcordia shows the apex beat to be changed in position and character, according to the relative degree of dilatation and hypertrophy of the left ventricle, and subsequently of the right ventricle.

First Stage.—In hypertrophy with slight dilatation (eccentric hypertrophy), the apex beat is displaced slightly to the left. The force is increased. In well-marked hypertrophy and dilatation the systolic impulse is lifting and heaving in character, and may jar the entire thorax and give a well-marked thrust if the ribs are elastic. Diastolic recoil may be detected, especially in cases where there is systolic retraction of the chest wall. When dilatation exceeds hypertrophy, the apex beat is diffused and lacks force. Diastolic thrill may be felt over the base of the heart or diffused over the entire præcordia. The pulse of aortic regurgitation is the most diagnostic of all the valvular diseases, and is also important in estimating the degree of the lesion. The characteristic feature of the pulse depends on the sudden emptying and collapse of the artery during diastole, followed by an equally sudden overfilling during systole, so that the highest and lowest points of tension follow each other very rapidly. The sphygmographic tracings (Fig. 42) show this. The passage of the pulse wave under the finger causes a sensation as of a series of hard particles passing, and the pulse is described as shotty or water-hammer (Corrigan's pulse). Elevating the arm intensifies this characteristic by favoring, through gravity, the emptying of the artery during diastole.

FIG. 66.



Double aortic murmur and mitral regurgitation. Second stage. Dotted lines show fluoroscopic outline of the heart.

The pulse is regular as long as hypertrophy compensates for the regurgitation. When it becomes unequal to this task, irregularity, both in rhythm and force, occurs. When the regurgitant stream is small, the water-hammer character of the pulse is not marked. It becomes progressively more prominent with increase in dilatation of the left ventricle, and it is also exaggerated when regurgitation is associated with anæmia or any condition favoring low resistance in the arteries and capillaries. Obstruction at the aortic orifice by interfering with the sudden emptying of the ventricle causes the pulse to be smaller and longer.

Second Stage.—With regurgitation at the mitral valve and hypertrophy of the right ventricle, the apex of the left ventricle is displaced from the chest wall. The impulse is carried downward, and also toward the median line, and may be felt in the epigastrium.

When regurgitation at the mitral valve takes place, the pulse becomes smaller; with the *third stage* failure of compensation, feeble and irregular.

Third Stage.—Failure of compensation and dilatation of the right ventricle cause a feeble, wavy, diffused impulse, which is also irregular in force and rhythm.

PERCUSSION.—The increase in the area of percussion dullness corresponds to the changes in the size and shape of the heart. *First Stage.*—When dilatation is slight and hypertrophy well marked, the area of dullness over the apex is sharply triangular, and extends downward and outward. Increase in dilatation causes the apex area to become rounded and extend further toward the left. There is no change along the left border of the sternum until, *second stage,* right ventricular hypertrophy occurs. *Third Stage.*—Dilatation of the right ventricle may cause dullness to be detected to the right of the sternum. Dilatation of the aorta may cause increased dullness at the level of the second interspace to the right of the sternum.

AUSCULTATION.—The murmur distinctive of regurgitation at the aortic orifice is diastolic, with the point of maximum intensity over the sternum or at the left edge opposite the third interspace and fourth costal cartilage, transmitted down the sternum to the ensiform cartilage, and at times to the apex. It is also heard over the aorta and arteries. (Fig. 54.)

The murmur varies in quality and loudness, point of maximum intensity, area of diffusion, and duration, according to the character and extent of the lesion at the aortic orifice and the secondary changes in the aorta and heart.

The quality and loudness of the murmur are dependent upon the nature of the opening through which the blood regurgitates (whether large or small, rough or smooth), and the force of the blood current determined by the blood pressure in the aorta.

First Stage.—The murmur may be soft, blowing in character, and scarcely audible. When the regurgitant stream is forced through a narrow, rough opening with great force, the murmur is harsh, grating, sawing and audible at times some distance from the patient. When the opening is large, the murmur is more gushing, and rapidly diminishes in loudness. When a murmur that has been loud becomes weaker and shorter, it indicates low pressure in the aorta, due to failure of cardiac power or decrease of resistance in capillaries.

The duration of the murmur and its relation to the normal cardiac sounds are important data in determining the nature and extent of the lesion. The longer the murmur persists during diastole, retaining the same degree of loudness, the smaller is the amount of blood that regurgitates through the valves and the slighter the effect on blood pressure in the aorta. Murmurs which are heard for a short time only during the early portion of diastole, and rapidly diminish in loudness, indicate free regurgitation.

The murmur may accompany, follow or take the place of the aortic second sound, according to the condition of the valves. When the incompetency is due to vegetations or deformities limited to one cusp, or when all the cusps are involved, but their elasticity is not destroyed, the aortic second sound may persist, and the murmur will accompany or follow it. In such cases the regurgitation is generally small, although free regurgitation may occur with persistence of the second sound.

When incompetency of the cusps is secondary to arterial disease or dilatation of the aorta, an accentuated aortic second sound may persist with the murmur. When the second sound is absent or the murmur replaces it, regurgitation is usually free, and not associated with stenosis.

The point of maximum intensity and the area of diffusion vary greatly. The murmur may be heard loudest in the aortic area, over the sternum, opposite the third interspace, at the ensiform cartilage, and at the apex, or it may be almost equally intense over the entire præcordia.

The point of maximum intensity is at the aortic area, and associated with persistence of the aortic second sound when regurgitation is secondary to thickening or atheroma of the aorta.

Free regurgitation, with very little diastolic tension of the aortic cusps, causes the murmur to be heard loudest over the right ventricle at the ensiform cartilage (conduction through the ventricular septum) and at the apex (conduction by the regurgitant stream).

Frequently the quality of the diastolic murmur heard at the apex differs from that over the sternum. The difference is due to the routes by which the vibrations made at the valve reach the surface; over the apex by the regurgitant stream giving a soft, blowing sound; over the sternum by the dense tissue giving a harsher, higher-pitched sound. (Fig. 55.)

Over the carotids and distal arteries, diastolic murmur is detected in proportion to the freedom of regurgitation. (Duroziez.)

A diastolic murmur is heard in addition to the normal systolic murmur (pressure murmur) when the stethoscope is firmly pressed on the artery. It never occurs except in free aortic regurgitation, and is dependent upon a strong reflux current toward the heart. In slight regurgitation it is absent.

In addition to the diastolic murmur, a systolic murmur is usually heard in the aortic area and over the arteries in well-marked cases of aortic regurgitation. The systolic murmur may be due to true obstruction or to dilatation of the aorta (relative obstruction), which occurs in a greater or less degree in aortic regurgitation.

When true obstruction and regurgitation are combined at the aortic orifice, the systolic murmur is harsh, and carried into the vessels of the neck with diminished intensity; but, in proportion to the obstruction, the carotid pulsations are less marked, and the pulse does not have the jerking, water-hammer character.

When the systolic murmur is due to dilatation of the aorta, without diminution in the size of the aortic orifice, it is soft, blowing at the base of the heart, and holds its intensity over the vessels, or may become louder; it is attended with marked pulsation in the carotids and the characteristic pulse. The diastolic murmur is relatively more intense than the systolic, and is especially intense over the carotids and distal arteries. The first sound of the heart is booming, and the pulmonic second sound is *not* accentuated.

Second Stage.—When incompetency occurs suddenly at the mitral valve, due to muscular weakness, the diastolic murmur at the base becomes less intense; the systolic murmur at the base becomes softer, and is not transmitted so far into the vessels of the neck. Over the apex another systolic murmur is heard, due to mitral regurgitation. It has all of the characteristics of that condition, and is associated with accentuation of the pulmonic second sound. After mitral regurgitation has occurred, with temporary relief of the left ventricle, recompensation may occur, with persistence of the mitral murmur. Increased power of the heart is shown by the returning harshness and loudness of both diastolic and systolic murmurs over the base of the heart.

Third Stage.—With failure of compensation by the right ventricle, the murmurs become indistinct or absent; there is a diminution of the pulmonic second sound, and the tricuspid regurgitant murmur and the physical signs of pulmonary congestion and œdema, already noted, supervene.

OBSTRUCTION AT THE PULMONARY ORIFICE OR IN THE PULMONARY ARTERY.

Obstruction of the pulmonary orifice occurring after birth, is one of the rarest of lesions, although as a congenital condition it is relatively frequent in proportion to the other cardiac defects, and is often associated with patent foramen ovale or other malformations of the heart. Although true obstruction is a most uncommon lesion, the diagnosis of obstruction at the pulmonary orifice is frequently made, as a number of conditions cause over the pulmonary valve area a systolic murmur whose points of maximum intensity and areas of diffusion are identical with those of stenosis.

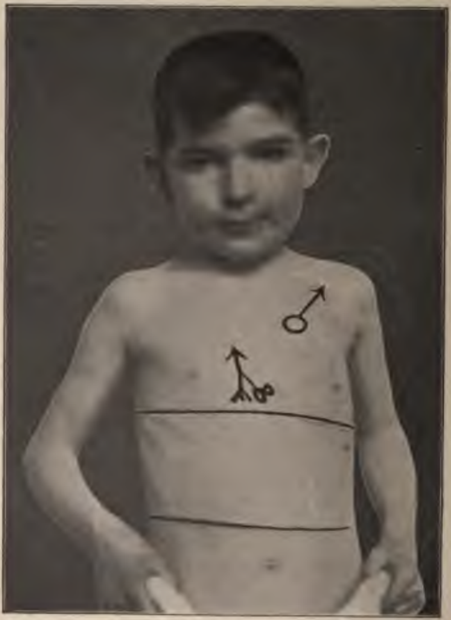
True obstruction at the pulmonary orifice may be due to changes in the cusps, as the presence of vegetations, thickening and adhesion of their borders, that produce a funnel-shaped opening; or the fibrous ring may be contracted.

The pulmonary artery may be narrowed just beyond the orifice by endarteritis. Pressure along the course of the pulmonary artery by pleuritic adhesions and mediastinal tumors (aneurisms, enlarged glands, malignant growths, etc.) may cause all the effects of obstruction at pulmonary orifice.

Non-obstructive conditions causing a murmur over the pulmonic area are (1) anæmic conditions, especially chlorosis; (2) neuro-cardiac diseases, as Grave's disease and allied disorders; (3) altered relation of the heart to the chest wall, due to displacement and uncovering of the heart by retraction of the overlying lung, pleuritic changes, etc.

Effect of Obstruction at the Pulmonary Orifice.—Interference with the flow of blood causes the right ventricle to become hypertrophied. If the obstruction is slight, no further effect may be produced. If the endocardial pressure is much increased during systole, regurgitation at the tricuspid valve occurs, causing both dilatation and

FIG. 67.



Pulmonary stenosis, tricuspid, regurgitation, pulsating liver.

hypertrophy of the right ventricle, interference with the general venous return and passive congestion of the abdominal organs, with œdema and effusion into the serous cavities. Clubbing of the fingers and toes, with enlargement and arching of the nails, occur in long-continued interference with return circulation.

Physical Signs. INSPECTION.—Cyanosis is almost always present. If the obstruction is congenital, it is most marked (blue baby). When the obstruction occurs in early childhood or later in life, cyanosis may only be noticed on exertion. Over the præcordia no change may be present unless there is dilatation and hypertrophy of the right ventricle, when the apex beat is carried downward, and is also seen in the epigastrium. With secondary tricuspid regurgitation, distension of the veins of the neck, and later pulsation in them, may be detected. The respiratory movements are increased, dyspnœa occurring on slight exertion.

PALPATION.—Over the base of the heart there is felt a distinct systolic thrill, with the point of maximum intensity at the left edge of the sternum opposite the second and third interspaces, which may be diffused over a wider area. The apex beat is most marked over the site of the right ventricle. The pulse is not affected until failure of the right heart, with tricuspid insufficiency.

PERCUSSION.—The area of cardiac flatness is slightly enlarged. When the dilatation of the right heart is marked, it may be detected to the right of the sternum.

AUSCULTATION.—The diagnostic sign is a harsh, *superficial,* systolic murmur, with the point of maximum intensity at the junction of the second rib with the sternum. It may be widely diffused over the upper portion of the chest, but is *never heard in the great vessels of the neck.* The second pulmonic sound is indistinct, or may be absent. The duration of the murmur is long, beginning sharply with systole and continuing almost to the second sound. The murmur is changed but slightly by the posture of the patient. Holding the breath causes it to become momentarily more intense, and then weaker. Forced inspiration intensifies it; forced expiration weakens or annihilates it. (Fig. 58.)

Non-Obstructive Murmurs in the Pulmonic Area.—These have the same time (systolic) and point of maximum intensity as the obstruction.

The *anæmic murmurs* are soft and blowing; they are rarely musical or harsh. They are markedly influenced by position, being weaker in the upright and louder in the recumbent postures. They are influenced in the same manner as are the structural murmurs by respiration. They are associated with arterial murmurs in the carotids and subclavians, or with a venous hum. The patient is pale. On exertion a ruddy color may occur, but no cyanosis. There is no change in the size of the right heart, and they are not accompanied by thrill. Frequently in women (for the first three days) after child birth a murmur may be heard over the pulmonic area. It is apt to have a scratching, crepitant-like quality. (Figs. 59-60.)

In the *neuro-cardiac* diseases the characteristics of the murmur are the same as those due to the anæmic. The cardiac action is increased, and there is marked pulsation in the vessels of the neck.

Altered Relation of the Heart to the Chest Wall.—Displacement of the heart by effusion into the pleural cavity frequently causes a murmur to be heard over the pulmonic area, which disappears on withdrawal of all or part of the fluid. Uncovering of the pulmonary artery by retraction of the lung also disturbs its relation, and causes a soft, blowing murmur to be present. These two conditions are associated with change in the pulmonary signs. Pleuritic adhesion, and retraction of the lung due to pleuritic or pulmonary changes, may cause slight narrowing of the pulmonary artery, as well as displacement. The murmur will be associated with depression of the thorax, with change in vocal fremitus, increased area of dullness opposite the fourth rib and signs of pulmonary and pleural changes.

Differential Diagnosis. *Systolic Murmurs.*—Aortic systolic murmurs usually have the point of maximum intensity to the right of the sternum, or may be localized in the pulmonary space. They are heard in the vessels of the neck. In aortic obstruction the pulse is small, and the cardiac change is limited during the earlier stages to hypertrophy of the left ventricle.

Aneurismal Murmurs.—Aneurism may produce a systolic murmur and thrill in the pulmonic area, but it will be attended with pulsations and increased dullness over the upper part of the sternum and pressure symptoms.

Patent Ductus Arteriosus.—The particular feature of the murmur of patent ductus arteriosus is its point of maximum intensity, which is further to the left of the sternum, and also it continues beyond the second sound. It is frequently associated with pulmonic obstruction as a congenital condition.

REGURGITATION AT THE PULMONIC ORIFICE.

Pulmonic regurgitation due to structural changes at the orifice is an extremely rare condition. When present, it is either congenial and associated with pulmonic obstruction; or, if acquired after birth, is due to ulcerative endocarditis or secondary to dilatation of the pulmonary artery. In the acquired form the cusps of the artery show pathological conditions analogous to those described under aortic regurgitation.

Functional or physiological incompetency of the pulmonary valves may occur as a result of dilatation of the pulmonary artery, due to high blood pressure secondary to mitral disease, or changes in the lung interfering with the pulmonary circulation, as emphysema, fibrosis, etc. Physiological incompetency may take place under excessive functional activity of the respiratory organs, as in contests of speed, endurance, etc. When it occurs under these conditions, it protects the integrity of the pulmonary circulation. The physical signs of this type of regurgitation are slight, and usually masked by the rapidity of the cardiac action and exaggerated breath sounds.

The Effect of Regurgitation at the Pulmonic Orifice.—The effect is to cause primary dilatation, with coincident hypertrophy of the right ventricle, and later tricuspid regurgitation, with its accompanying phenomena.

Physical Signs. INSPECTION.—The apex beat is carried to the left. Pulsations may be seen in the epigastrium and in extreme dilatation of the heart to the right of the sternum. Distension of and pulsations in the veins of the neck occur with tricuspid regurgitation.

PALPATION.—The apex beat is diffuse, and most marked over the right ventricle and close to the sternum and in the epigastrium. Over the base of the heart a diastolic thrill may be felt. Cardiac dullness is increased, and may be detected beyond the right edge of the sternum over the site of the pulsation.

AUSCULTATION.—A diastolic murmur is heard with the point of maximum intensity in the second left intercostal space, with the area of diffusion downward along the sternum and to the apex of the right ventricle. The murmur may be harsh, rasping and superficial, especially when associated with pulmonary obstruction, or soft and blowing when due to dilatation of the pulmonary artery. (Fig. 61.)

Differential Diagnosis.—Pulmonic regurgitation is easily distinguished from aortic regurgitation when the two murmurs have their usual point of maximum intensity and area of diffusion. However, they may coincide in these respects. In pulmonic regurgitation the signs of hypertrophy and dilatation are limited to the right side of the heart; in aortic regurgitation the left ventricle is chiefly involved. In pulmonic regurgitation, dyspnœa and cyanosis, with clubbing of the fingers, are marked, also pulsations are noted in the veins. In aortic regurgitation the pulsations in the carotids and character of the pulse are distinctive. In pulmonic regurgitation a murmur is *not* heard in the vessels of the neck; in aortic regurgitation a double murmur is heard in the vessels of the neck and over the arteries.

OBSTRUCTION AT THE TRICUSPID ORIFICE.

Obstruction at the tricuspid orifice is one of the rarest cardiac lesions. Most of the cases are not recognized during life. Of the recorded cases, nearly 80 per cent. occurred in women.

The obstruction is usually due to adhesions of the free margins of the leaflets, converting the valve into a funnel-shaped structure, or reducing the opening to a mere slit.

Effect of Tricuspid Obstruction.—The effect varies according to the degree of obstruction, and may be limited to the right auricle or extend beyond to the venous circulation. When the stenosis is slight, hypertrophy of the right auricle is frequently sufficient to overcome the obstruction to the flow of blood through the orifice. When the power of the auricle is insufficient, interference with the return circulation occurs, causing congestion of the abdominal organs and effusion into the serous cavities.

Physical Signs.—The physical signs present in tricuspid obstruction vary greatly. In some of the reported cases they were well marked, and in others indistinct. As in mitral obstruction, the murmur may be present at one time and absent at another.

INSPECTION.—When the obstruction is slight and is compensated for by the right auricle, no change is noted. Interference with venous circulation is shown by distension of the superficial veins, especially of the neck. When pulsations occur, they are synchronous with the auricular systole. There is also some duskiness of the skin and œdema. Effusion into the pleural cavity may cause change in the position of the apex beat.

PALPATION.—At times a thrill is felt. Its maximum intensity may be over the valve along the left border of the sternum, or at the apex of the right ventricle in the fifth interspace. It may occur in both locations at the same time with nearly equal intensity, or its maximum intensity may alternate, being first in one place, then in another. The radial pulse shows no change, except in marked obstruction, when it is rapid, irregular, small and compressible. Cardiac impulse may be normal or in marked obstruction, feeble and irregular.

PERCUSSION.—Very little change is produced, except that dilatation of the right auricle may increase the transverse area of percussion flatness at the level of the fourth rib.

AUSCULTATION.—The diagnostic sign of tricuspid obstruction is a pre-systolic murmur, with the point of maximum intensity over the lower portion of the sternum at the upper edge of the ensiform cartilage. Its area of diffusion corresponds to the area of superficial cardiac dullness. The murmur may occupy the entire diastole, with accentuation just before the systolic impulse. (Fig. 56.)

Differential Diagnosis.—The distinctive features of tricuspid obstruction are absence of ventricular hypertrophy, marked venous turgescence, the presence of a thrill and a pre-systolic murmur limited to the area occupied by the right ventricle. On account of its frequent occurrence with mitral obstruction, it is masked by the signs of that lesion.

MITRAL OBSTRUCTION.—Mitral obstruction has also a pre-systolic murmur, and inspection shows right ventricular hypertrophy, and, when venous overfilling is present, also hypertrophy and dilatation of the right ventricle.

The thrill is more vibratory in quality, and is located at the apex, but its termination by ventricular systole is more definite. The murmur, although it may be heard during the major part of the diastole, has a pre-systolic accentuation more marked, and the murmur terminates with the short, intensified first sound. The maximum intensity is at the apex, but it is not diffused widely, and is never heard along the left border of the sternum or over the ensiform cartilage (tricuspid area).

The pulmonic second sound is accentuated in mitral obstruction, and indistinct in tricuspid obstruction.

REGURGITATION AT THE TRICUSPID ORIFICE.

Regurgitation at the tricuspid orifice occurs under two separate conditions. (1) It may be due *(a)* to vegetations on the leaflets, preventing perfect coaptation, *(b)* to thickening, shrinkage or deformity of the leaflets, *(c)* to induration and shortening of the chordæ tendineæ and papillary muscles. These structural changes are usually due to endocarditis and pericarditis which are likely to involve other valves besides, and are relatively rare at the tricuspid valve alone.

(2) Incompetency at the tricuspid orifice is most frequently dependent on dilatation of the right ventricular cavity and the auriculo-ventricular ring, which becomes too large to be closed by the valves. Relative valvular insufficiency occurs much more readily at the tricuspid valve than at the mitral. There is abundant proof that there exists a safety valve action at the tricuspid orifice, and that slight temporary regurgitation occurs whenever the pressure in the pulmonary artery reaches a certain height. Under normal conditions, the functional or physiological regurgitation that occurs under extreme exercise or exertion is temporary, disappearing with the return of the blood pressure to normal. When tricuspid incompetency from dilatation of the right ventricle is secondary to persistent high

pressure in the pulmonary artery or obstruction at the pulmonary orifice, it is permanent, and represents another broken link in the chain of cardiac compensation.

The cause of high blood pressure in the pulmonary artery may be obstructive disease in the lungs (emphysema and fibroid changes) or lesion at the mitral orifice, either primary or secondary (see Mitral Regurgitation and Obstruction). When the relative insufficiency is due to transient increase of pressure in the pulmonary artery (as in acute bronchitis, overexertion, etc.), or to temporary disturbance of cardiac nutrition (pyrexia, toxines, anæmia, etc.), the dilatation may become less and the valves again become competent to close the orifice with the disappearance of the disturbing factors.

Effect of Tricuspid Regurgitation.—The immediate effect is upon the right auricle, which may become dilated to an enormous extent. The secondary hypertrophy is never very marked, and is always inadequate to compensate for the lesion. The great veins become dilated, the inferior vena cava more so than the superior. The valves protecting the veins at the root of the neck become incompetent. Venous stasis causes general œdema. One of the earliest effects of tricuspid regurgitation is enlargement of the liver and the occurrence of ascites and hydrothorax.

Physical Signs. INSPECTION.—As an isolated lesion, the apex beat is displaced inward toward the median line, and may be beneath the sternum or in the epigastrium. Impulses due to auricular systole may be seen in the third and fourth interspaces to the right of the sternum. When secondary to left-side cardiac disease, the apex is displaced downward and outward, and also is seen in the epigastric region. When secondary to emphysema, distension of the lung causes the epigastric pulsation to be specially prominent. Cyanosis is usually present. In long-continued interference with return circulation clubbing of the fingers occurs.

Systolic venous pulsations are present in the jugulars, most marked on the right side. In marked cases pulsation of the liver may be seen. The respiratory movements are increased unless hydrothorax occurs, when there will be loss of motion over the lower portion of the thorax.

PALPATION.—Cardiac enlargement corresponds to that noted by inspection. When tricuspid regurgitation is a primary disease, the epigastric pulsations are weak and undulatory. When secondary to cardiac or pulmonary changes, the pulsations are more forcible and heaving. When the ear is placed over the tricuspid area, a characteristic wavy impulse is detected in addition to the murmur. Hepatic pulsations are one of the diagnostic signs, and must be differentiated from impulse transmitted to the liver from the aorta or the enlarged right ventricle. Pressure on the liver in tricuspid regurgitation increases jugular pulsations.

PERCUSSION.—The cardiac area is enlarged to the right. Dullness may be detected beyond the right border of the sternum at the level of the fourth rib.

AUSCULTATION.—The murmur of tricuspid regurgitation is systolic, soft, blowing, with the point of maximum intensity along the sternum at the junction of the fifth and sixth costal cartilages. The area of diffusion corresponds to the extent of the præcordia. It does not extend above the third rib nor beyond the apex. The intensity of the murmur is increased during expiration, and is diminished or absent during inspiration. The second sound varies. When tricuspid regurgitation is primary, it is weak and indistinct. When secondary to increased pressure in the pulmonary artery, the second sound is accentuated. (Fig. 57.)

Differential Diagnosis.—The distinctive features of tricuspid regurgitation are extension of the cardiac area to the right, systolic pulsations in the veins and liver, and a soft, blowing, systolic murmur, with the point of maximum intensity over the site of the tricuspid valve and heard over the præcordia. It is to be differentiated from other lesions characterized by a systolic murmur.

Mitral Regurgitation.—In mitral regurgitation the murmur is heard with the point of the maximum intensity at the apex, and it is carried *beyond* the præcordia to the left into the axilla, and that it is heard behind. The thrill is vibratory and distinctly presystolic. The apex is displaced to the left and downward.

Aortic Stenosis.—The systolic murmur is heard above the third rib, and is carried up into the vessels of the neck. It is also associated with characteristic pulse.

Pulmonic Obstruction.—In pulmonic obstruction the point of maximum intensity and the area of diffusion are diagnostic. (Fig. 58.)

DISEASES OF THE MYOCARDIUM.

Hypertrophy and Dilatation.

Hypertrophy of the cardiac muscle may occur in two forms[1]—(1) simple hypertrophy, (2) hypertrophy with dilatation of the cavity, or eccentric hypertrophy. It may be limited to the walls of a single cavity, or may be general. Cardiac dilatation is also divided into two forms[2]—(1) simple dilatation, in which there is increase in the size of the cavity, with thinning of the wall; (2) dilatation with hypertrophy, in which, with enlarged size of the cavity, there is increase in the relative thickness of the heart wall. In both eccentric hypertrophy, and dilatation with hypertrophy we have increase in the size of the cavity, with increase in thickness of the heart muscle. The distinction between these two conditions depends upon the relative efficiency of the cardiac muscular power to the dilatation. When the working power of the heart is increased beyond what is needed to overcome the cause and effect of the dilatation, it is classified as eccentric hypertrophy. When, on the other hand, dilatation is predominant, and the increased muscular tissue barely compensates for the dilatation, it is classified as dilatation with hypertrophy.

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[1] Concentric hypertrophy characterized by increase in thickness in the walls of the cavity with diminution of its size is rarely recognized during life.

[2] Atrophic dilatation has been described dependent on wasting of the cardiac muscle coincident with increase in size of the cavity.

Physical Signs.—Cardiac dilatation and hypertrophy are generally secondary conditions.

The physical signs present in the different forms are fully described under "Inspection," "Palpation," "Percussion" and "Auscultation," also under the diagnosis of the different valvular diseases.

Acute Myocarditis.

This may exist in two forms, (1) as an acute degeneration of the muscle tissue of the heart dependent upon infectious fevers (cloudy swelling, parenchymatous myocarditis); (2) as an inflammatory affection of the interstitial tissue of the heart, which may also be due to infectious diseases, or may be secondary to acute inflammations of the pericardium or endocardium.

Localized or suppurative myocarditis may result from infection from micro-organisms, secondary to suppurations in distant portions of the body or due to extension from infectious endocarditis.

Physical Signs.—The physical signs present are those indicative of defective muscular power, with varying degrees of dilatation. The earliest change noted is feebleness of the cardiac impulse, both to inspection and palpation. Auscultation shows gradual diminution in the muscular element of the first sound, with accentuation of the aortic and pulmonic second sounds. When dilatation occurs, the murmurs of mitral and tricuspid regurgitation may be present.

Chronic Myocarditis.

Chronic changes in the myocardium may be due to (1) fatty degeneration of the muscle tissue; (2) fibroid degeneration (fibroid or interstitial myocarditis); (3) fatty overgrowth (fatty infiltration or cor adiposum).

Physical Signs.—The physical signs in disease of the myocardium are those of weak heart, "weak in muscle power and weak in its resistance to blood pressure;" signs of hypertrophy and dilatation are usually present in varying degree, according to the cause of the condition.

INSPECTION.—The apex beat of the heart may be in the normal position, or displaced; when the changes in the myocardium are secondary to sclerosis or atheroma in the blood-vessels, the apex beat will be displaced outward and to the left in proportion to the hypertrophy and dilatation. With insufficiency of cardiac power, the patient shows disturbances of the superficial circulation and slight blueness of the extremities. Atheroma of the blood-vessels is shown by prominence of the temporal and other arteries. In fatty overgrowth there is associated obesity.

PALPATION.—The location of the impulse depends upon the presence of hypertrophy and dilatation. In fatty degeneration the impulse is feeble and diffused. When associated with hypertrophy, especially of the fibroid type, the impulse, while heaving, lacks the lifting force proportionate to the size of the heart. The pulse is weak in comparison to the seeming effort of the heart. When cardiac power is insufficient, arrhythmia occurs, which may be constant or induced by slight exertion.

PERCUSSION.—The dimensions of the heart are increased with hypertrophy and dilatation in all directions. When dilatation is predominant, percussion dullness extends above the third interspace and to the nipple line or beyond it. In fatty overgrowth the percussion area of the heart is increased. Frequently it is impossible to map out the heart on account of the thickness of the superficial tissue.

Auscultation.—The characteristic sign is a short, feeble first sound at the apex, with a relatively louder second sound over the aorta. The pulmonic second sound is not increased in intensity. Usually there is reduplication of the first sound. With the occurrence of dilatation and valvular incompetency, soft, blowing murmurs are heard in the mitral and tricuspid areas.

Diagnosis of myocarditis rests upon the above physical signs, associated with other evidence of insufficiency of muscular power, as dyspnœa, oppression in the chest, or anginal pain on exertion and after meals, syncope with cardiac irregularity.

CHAPTER XIII.

DIAGNOSIS OF THE DISEASES OF THE PERICARDIUM.

The smooth surfaces of the pericardium, lubricated by the normal secretion of the serous membrane, glide over each other during the cardiac and respiratory movements without producing any vibrations which can be detected by palpation or heard as a friction sound. The amount of secretion contained in the pericardial sac in health varies within very narrow limits, and is never present in such quantity as to be detected by physical examination. Pericardial diseases produce distinctive physical signs, according as the normally smooth surfaces are altered so as to produce vibrations which may be felt or heard, or the sac is distended by fluid.

Pericarditis.

According to the nature of the changes in the pericardial sac, three forms of pericarditis can be recognized by physical signs, irrespective of their ætiology. The morbid conditions of each form may exist separately or may be combined in varying degrees, and due to widely differing causes. As a number of non-inflammatory conditions of the pericardium may produce changes similar to those due to the inflammation, and as the physical signs depend upon the altered state of the sac, they will be described with the different forms of pericarditis.

(1) **Dry, Plastic or Fibrinous Pericarditis.**— In this form the surfaces may be abnormally dry from diminished

secretion, dependent upon loss of blood or fluid from the body, or changes in the serous membrane during the early stage of inflammation. Later during the inflammatory attack the surfaces may be covered with thick, tenacious exudate (fibrinous or plastic). The surfaces may also be roughened by fibrous, tubercular or malignant growths. The physical signs will vary according to the nature of the change.

(2) **Effusion into the Pericardium, Pericarditis with Effusion.**—The fluid poured into the sac may be *sero-fibrinous, serous, purulent* or *hemorrhagic.* Serous effusion, small in amount, may coincide with a fibrinous exudate which coagulates on the surfaces, while the more liquid element gravitates to the bottom of the sac (sero-fibrinous pericarditis). The liquid element (serum) of the inflammatory exudate may be excessive and rapidly fill the pericardial sac without the occurrence of the dry, or plastic stage. Serous effusion into the pericardium occurs as part of a general dropsy and interference with the return circulation (hydro-pericardium), and is unattended with any inflammatory changes. It usually accompanies effusion into other serous sacs.

Purulent effusion may occur primarily, as in septic disease, or be due to secondary changes in sero-fibrinous or serous inflammatory pericarditis. Hemorrhagic effusion (hæmo-pericardium) may be inflammatory or be dependent upon new growths, blood condition (scurvy, purpura), rupture of the heart or of a cardiac aneurism.

The physical signs present in effusion are in proportion to the amount of fluid and the effect of the enlarged pericardium upon the heart and surrounding organs. The nature of the fluid does not modify the physical signs.

(3) **Pericardial Adhesion. Adherent Pericardium.**—This is usually secondary to inflammatory diseases of the pericardium. It may be limited to a few bands loosely stretched between the two surfaces and not interfering

with the cardiac action; or the two surfaces may be closely united, or the sac may be completely obliterated, in which case the movements of the heart will be entirely or in part restricted and secondary hypertrophy or dilatation with hypertrophy will occur.

Physical Signs.—As the three forms of pericarditis may be variously combined, or follow each other during the course of an attack of pericarditis, they will be considered together.

INSPECTION. *Dry, Plastic or Fibrinous Form.*—There is no change in the contour of the præcordia. The apex beat is in the normal position, the impulse more forcible, due to cardiac irritability. When associated with myocarditis, the impulse is weaker and more diffused.

Second Form. Effusion into the Pericardium.—Change in the præcordia will be in proportion to the quantity of fluid and the elasticity of the chest walls. The effects on the shape and size of the thorax will be most noticed in thin persons and in children, and may be masked in stout persons. The apex beat is seen at a higher point on the chest wall, according to the amount of fluid present. This is not due to elevation of the anatomical apex, but to the changed relation of the anterior portion of the heart to the chest wall, so that a point nearer the base of the heart strikes the chest wall and gives the visible and palpable apex beat. *(a)* Small effusions may produce no change in the præcordia. The apex beat is displaced inward and slightly elevated. *(b)* In medium-sized effusions the intercostal spaces along the left side of the sternum, from the third to the sixth, are filled out, and may be slightly bulging. The thorax over the apex is slightly enlarged. *(c)* In large effusions the bulging over the præcordia is marked, and the lower end of the sternum is pushed out; the epigastric region is more prominent, the costal cartilages and ribs are elevated, and the intercostal spaces on both sides of the sternum are wider, the normal depressions being absent.

The changes in the lower portion of the chest are due to diminished negative pressure within the thorax and depression of the diaphragm and liver by the weight of the fluid. The visible impulse is usually seen above the fourth rib. In extreme distension pericardial bulging may extend as far as the second rib.

Enlargement of the pericardial sac causes reduction of the negative pressure within the chest, and allows of the elevation of the bony thorax and changed relation between the ribs individually, and especially between the first rib and the clavicle. Ewart has called attention to the changed relation between the clavicle and the first rib (first rib sign) as important in diagnosis of effusion of considerable amount. The left clavicle is raised to a higher level, so that the upper edge of the first rib on the left side can be felt as far as its sternal attachment. When the amount of fluid is sufficient to produce a positive pressure, instead of the normal negative pressure in the pericardium, cyanosis occurs, also pulsations in the veins of the neck, due to auricular contraction.

The effect of posture on the apex beat in effusion is the opposite of what occurs in other conditions, causing an elevation of the apex beat. When the patient is on the right side, the apex beat becomes lower and more distinct; in other conditions it is higher, weaker or absent.

Adherent Pericardium.—Flattening of the chest over the lower portion of the præcordia usually occurs. Occasionally there is bulging, due to hypertrophy and dilatation of the heart. Systolic retraction of the chest wall may be limited to the apex and the intercostal spaces to the left of the sternum, or to the left half of the epigastrium. The respiratory movements are altered when extensive pericardial changes are present. With inspiration there is loss of expansion over the lower portion of the left chest below the horizontal nipple line, and drawing in of the end of the sternum. Protrusion of the abdomen, and frequently ascites, may occur. The apex beat is displaced outward and downward or elevated as high as the fourth rib, according as the heart is hypertrophied or dilated or fixed by adhesions in an abnormal position. The apex beat does not vary with change in the posture of the patient. The diastolic rebound of the heart may be visible. Diastolic collapse of the veins is occasionally seen.

PALPATION. *Dry, Plastic or Fibrinous Pericarditis.*—The cardiac impulse may be increased. Friction fremitus is felt over different portions of the præcordia in a small proportion of cases; it is most readily detected by making moderate pressure in the intercostal spaces with the finger tips. The rhythm of the fremitus generally corresponds with the systole, but may extend into the diastolic period or be confined to it. It does not have a well-defined or stable point of maximum intensity, but varies in different cases and in the same case at different times, although it is more frequent and persistent over the base of the heart. The area over which it is felt is usually the circumscribed point of maximum intensity; occasionally it is felt over the entire præcordia, and may be perceptible in widely separated spots. The fremitus gives the sensation of being superficial and produced by the rubbing together of dry, rough or sticky surfaces. In acute cases the friction fremitus is present for a short time only, and varies from day to day in situation, extent and character.

Effusion into the Pericardium.—Friction fremitus, usually absent over the lower portion of the præcordia, may be detected above the fourth rib and over the site of the great vessels. Cardiac impulse is felt at a higher level, and is more diffused according to the amount of diffusion. Fluctuation over the præcordia may be present when the amount of fluid is sufficient to give positive pressure in the pericardium. Diminution in the amount of fluid causes the cardiac impulse to be felt at a lower point, and to become more defined. Removal of the fluid is frequently attended with reappearance of friction fremitus.

Adherent Pericardium.—In addition to the abnormal movements of the chest wall noted in inspection, the cardiac impulse frequently gives the sensation of being abnormally close to the chest wall. According to the situation and extent of the adhesions and the secondary changes in the heart (hypertrophy or dilatation), the impulse will be displaced to various portions of the præcordia, and will be strong, diffused or of a more undulating character. When adhesions unite the heart with the chest wall, diastolic shock is a marked symptom.

PERCUSSION. *Dry, Plastic or Fibrinous Pericarditis.*—No change in the area of superficial flatness or relative dullness is detected unless myocarditis with dilatation is present. The extreme edge of cardiac dullness corresponds closely to the apex beat.

Effusion into the Pericardium.—The most distinctive sign of pericardial effusion is change in the extent and outline of the area, of cardiac flatness and dullness and their relation to each other. These changes can be detected by *careful* and *systematic* percussion when but a small quantity of fluid is present. The area of flatness increases, while that of relative dullness diminishes. As the pericardium increases in size, change in the interthoracic pressure (negative pressure) allows that portion of the lungs which normally covers the pericardium to retract, so that there is not the gradual change from normal pulmonary resonance to cardiac flatness. In extreme distension of the pericardium, the change in the intrathoracic pressure may be sufficient to cause diminution of function in different portions of the lungs, with characteristic pulmonary resonance. A small effusion causes change in the percussion area, most marked at the level of the fifth rib. *(a)* To the left the flatness extends to the site of the normal apex beat or slightly beyond. The area of relative dullness in this region is decreased. *(b)* At the right of the sternum dullness is detected in the fifth intercostal space (cardio-hepatic triangle), and the cardio-hepatic triangle becomes more obtuse (Rotch's sign). As the fluid increases in amount, the area of flatness extends in all directions, especially laterally, at the lower level of the pericardium. With the increase in extent, the contour of the pericardial flatness also changes, becoming more pyramidal, with the two sides meeting the base at nearly equal angles. More marked distension causes the lower segment of the pericardium to be more globular and the upper portion more cylindrical, so that the typical pear-shaped outline obtains. Sternal resonance is altered by the effusion. As the sac becomes filled, the resonance becomes impaired, and is later replaced by flatness. The change in loss of resonance occurs from below upward, in contradistinction to that occurring in aneurism of the aorta. The percussion note over the lung is changed according to the amount of fluid present. As the fluid increases, the enlarged pericardium allows retraction in the overlapping lung and diminution of tension, so that the normal pulmonary resonance is replaced by a tympanitic quality (Skoda's resonance), which in large effusion may be present over the apex of the lung on the left side.

"Whenever fluid is effused into the pericardium, the normal resonance is modified at the left posterior base in the most definite way. A patch of marked flatness is found at the left inner base, extending from the spine for a varying distance outward, usually not quite so far as the scapular line, and ceasing abruptly with the vertical boundary. Above, its extension is also variable, according to the size of the effusion. Commonly it does not extend higher than the level of the ninth or tenth rib, and here, again, its horizontal boundary is abrupt. Its shape is that of a square, and is quite unlike that of any dullness arising from pleuritic effusion." (Ewart.)

Over the liver, the upper limit of both dullness and flatness is lowered; the lower edge of the liver is below the free border of the ribs in proportion to the depression of the diaphragm.

Adherent Pericardium.—Changes in thickness of the pericardium do not affect the percussion area. Any alteration in size or shape is due to the presence of hypertrophy or dilatation of the heart.

AUSCULTATION. *Dry, Plastic or Fibrinous Pericarditis.*—The diagnostic sign of this form is the friction sound. The quality of the friction sound varies according to the condition of the pericardial surfaces. It may be soft and murmurish, simulating the endocardial murmur; soft, dry, grazing or brushing, as when the dry finger tips or pieces of silk are gently rubbed together close to the ear; sticky; or harsh, creaking or grating, as when new leather is bent. In addition to the friction quality, the sensation of being superficial is a distinctive feature. The rhythm of the friction sound, while dependent upon cardiac movement, is not accurately limited to the periods of the normal cardiac sounds, but by extending beyond them it gives a double or to-and-fro rhythm, which is characteristic of pericardial friction. The point of maximum intensity is not stable, and the sound is not conducted beyond the præcordia except in rare cases, when the conduction is due to change in the surrounding lung tissue.

The friction sound may be heard over any portion of the præcordia, but its most persistent site is over the base of the heart, opposite the third and fourth ribs on the left side. The intensity of the sound is influenced by *(a)* pressure, *(b)* position, and *(c)* respiratory movements in a rather characteristic way. *(a)* Pressure over the præcordia causes the friction sound to be heard, or, when present, to become more intense and audible over a wider area and for a longer period of time; it also increases the to-and-fro character, and emphasizes its want of synchronism with the normal cardiac sounds. *(b)* Posture. Bending the body forward, so that the heart comes in contact with the anterior portion of the pericardium, affects the friction sound in the same manner as pressure. It intensifies the sound, and increases the area over which it is heard. *(c)* Respiratory movements. The intensity, quality and extent of the friction sounds change during inspiration and expiration. In some cases the increase in these features occurs during inspiration, and at others during expiration. The significant fact is that the sound *is* altered to a marked degree during either inspiration or expiration. Variability from day to day in the site, rhythm, intensity and quality is a distinctive feature of pericardial friction sounds.

Effusion into the Pericardium.—As fluid accumulates, there is disappearance of the friction sound, as there was of friction fremitus, over the lower portion of the pericardium except when separation of the anterior surface of the heart from the pericardium is prevented by adhesions. At the base, and especially where the pericardium is reflected along the great vessels, the friction sounds may persist during the entire course. The cardiac sounds over the site of the normal apex are weak or inaudible, and are more distinctly heard toward the base of the heart over the sight of the visible impulse. The pulmonic second sound may be increased.

The respiratory sounds are also modified. The respiratory murmur and vocal fremitus along the normal site of the anterior border of the lung, especially on the left, are feeble or absent. Bronchial breathing and ægophony may be heard in large effusions below the right nipple and behind at the angle of the left scapula.

If the patient lies with face downward, or leans forward on the knee and elbow, the dullness and bronchial breathing in the back disappear. With absorption of the fluid, the friction sound may return, and there will be early disappearance of the respiratory signs.

Cardiac sounds are heard in the normal site and with normal intensity when myocardial changes have occurred.

Adherent Pericardium.—The auscultatory signs are not distinctive, and depend on the condition of the surfaces and the degree of change produced in the heart and at the valvular orifices. A rough pericardial friction sound may be heard over different portions of the præcordia, especially the base. The cardiac sounds may vary greatly. The first sound may have the valvular quality accentuated or may be doubled; the second sound may be reduplicated and accentuated. Both sounds may be heard abnormally clear behind. Endocardial murmurs, due to dilatation of the cavities may be present.

Differential Diagnosis.—The distinctive features of acute, dry, fibrinous pericarditis are friction fremitus and friction sound of marked superficial, rubbing quality, with no fixed point of maximum intensity. The sound does not coincide with the areas of the normal valve sounds nor with the areas of endocardial murmurs, and it is not transmitted beyond the præcordia. Its rhythm is not accurately synchronous with the periods of the cardiac sounds, but has a double, to-and-fro character. The intensity of the sound varies during the respiratory movements, and is increased by pressure over the præcordia, and is loudest when the patient is erect or bending forward. There is no evidence of cardiac enlargement.

Acute Endocarditis and Chronic Valvular Disease.—The thrill present in endocardial diseases is localized at the apex (mitral obstruction) or at the base (aortic stenosis or regurgitation). It has a definite relation to the rhythm of the cardiac sounds, and does not give the impression of being superficial. The endocardial murmurs have a definite area of maximum intensity and area of diffusion. Murmurs made at certain valves are heard beyond the præcordia. The intensity of the murmur is not altered by pressure. Systolic murmurs are loudest in the recumbent posture and fainter in the erect. In chronic valvular disease, hypertrophy and dilatation of the heart are present.

Pleuritic Friction and Pleuro-pericardial Friction Sounds.—Friction sounds made in the pleura by the movement of the heart may simulate those of pericarditis. They are distinguished by the effect of respiration on the intensity and quality of the sound. On deep inspiration, the sound is intensified. When the patient takes a full breath and holds it, the sound disappears.

EFFUSION INTO THE PERICARDIUM.—The distinctive features of effusion into the pericardium are that the visible cardiac impulse is raised; the lower portion of the præcordia is fuller than normal; the intercostal spaces are wider and smoothed out, or even bulging, according to the extent of the effusion, and pulsations may be noted above the fourth rib. The percussion area of cardiac flatness is increased, and extends to the left beyond the normal site of the apex beat. To the right there is at first dullness in the fifth interspace, with gradual extension to the right of the sternum; the area of relative deep-seated cardiac dullness is diminished, with a sharp line of demarcation between pulmonary resonance and cardiac flatness. The outline of the cardiac flatness becomes more pyramidal or pear-like. The sternal resonance is impaired from below upward in proportion to the distension of the pericardium. On auscultation, the cardiac sounds are feeble or absent over the apex, but are heard more clearly toward the base. Friction sounds over the base of the heart may be sometimes detected. Over the lung, the normal vocal fremitus and respiratory murmur are absent over the normal position of the anterior border. In extensive effusion areas of bronchial breathing are found in the right intermammary region in front, and at the angle of the left scapula behind.

Dilatation and Hypertrophy of the Heart.—The visible, palpable apex beat is carried downward and to the left. Left ventricular hypertrophy causes forcible apex beat; in right ventricular hypertrophy the apex beat is diffused, and is seen and felt also in the epigastrium. When dilatation predominates, the impulse is feeble and diffused, but felt at the lower portion of the præcordia and in the epigastrium.

Dullness does not extend beyond the apex beat to the left, nor flatness beyond the right of the sternum. The normal sternal resonance is but slightly impaired. There is relative greater increase in the area of dullness over that of flatness.

The cardiac sounds are heard distinctly at the apex. When hypertrophy and dilatation are due to valvular lesions, the characteristic murmurs are present.

Effusion into the Left Pleura.—In this condition displacement of the heart to the right gives elevation of the impulse and dullness or flatness to the right of the sternum. But the flatness on the left side extends beyond the præcordia, and has its highest point in the axillary line, and is noted behind. Bronchial breathing, when present, is above the level of the fluid, but not at the angle of the scapula.

ADHERENT PERICARDIUM.—The characteristic features of adherent pericardium are flatness of the chest over the lower portion of the præcordia, with systolic retraction over the apex or other portions of the præcordia, according to the seat of adhesion, associated with diastolic rebound, and at times diastolic collapse of the veins. Rough, creaking pericardial friction sounds may be present.

There are no pathognomonic signs of this condition, and diagnosis rests on the relation of the physical signs to each other.

Cardiac Hypertrophy and Dilatation.—Systolic retraction over the apex may be present in massive cardiac hypertrophy with adhesions between the pericardial sacs. It is distinguished from adherent pericarditis by the history of the case, and the presence of valvular lesions causing the hypertrophy.

Pneumo-pericardium.

Gas in the pericardium may be due to the decomposition of the fluid contents or to perforation of the pericardium, which may be traumatic or may be due to ulceration from air-containing spaces.

Physical Signs. INSPECTION.—Distension of the pericardium causes bulging over the præcordia. The apex beat is weak or absent, and is most marked when the patient bends forward.

PALPATION.—Emphysematous crepitations may be present. Succussion splash may be felt with movement of the heart when air and fluid are present.

PERCUSSION.—The percussion sounds are most distinctive. Over the pericardium there is a tympanitic percussion note, frequently with a ringing, metallic quality. Cracked-pot resonance may be present on forcible percussion when there is free opening. The percussion note over the præcordia varies with the posture of the patient. In the recumbent posture the entire præcordia is resonant. When the patient stoops forward, bringing the heart against the chest wall, a small zone of dullness may be detected. When fluid and air are in the pericardium, flatness may be present over the lower portion of the pericardium and tympanitic resonance above. The line of flatness changes with the position of the patient.

AUSCULTATION.—The character of the sounds vary according to the presence of air alone or of air and fluid in the pericardium. With air alone, or if there is but little fluid, the heart sounds have a loud, ringing, metallic quality. When the cardiac action produces movement of the fluid, metallic splashing or churning (water wheel) sounds are present. Endocardial murmurs, when present, are intensified by reverberation in the pericardium.

CHAPTER XIV.

DIAGNOSIS OF DISEASES OF THE BLOOD-VESSELS.

ANEURISM OF THE AORTA.

Dilatation of the aorta occurs in two forms: (1) Fusiform or cylindrical. In this form, at the seat of the dilatation, there is increase of the lumen of the artery in all directions without the occurrence of pouching at any point, and unattended by the formation of any tumor or by pressure symptoms, even when the other physical signs of dilatation are very marked. To this form belong those general dilatations of the aorta and of the arteries given off from the arch, which may be due to changes primarily in the blood-vessels (aortitis, atheroma, etc.) or secondary to regurgitation at the aortic orifice. This form persists for a long time in a stationary condition, and beyond pulsation over the course of the artery, and the thrill and the murmur due to the enlarged lumen of the tube gives rise to no secondary symptoms.

Acute dilatation of the blood-vessel frequently occurs in inflammatory conditions of the aorta. It may disappear under rest and treatment, or may remain as a permanent dilatation.

(2) Sacciform or circumscribed form. This is due to dilatation involving a part only of the circumference of the wall of the artery, with the formation of a circumscribed tumor whose cavity is connected with the lumen of the artery by an opening of varying size, but the

diameter of the opening is smaller than that of the aneurismal sac.

The aneurismal tumor, filled with coagulated or fluid blood, produces physical signs and symptoms which are in part due to the tumor itself and in part dependent upon its pressure upon the bony thorax, the heart, œsophagus, trachea, bronchi, lungs and mediastinal structures. The signs and symptoms of aneurism vary according to the size and situation of the tumor and the direction of its pressure.

TABLE OF PRESSURE SIGNS IN ANEURISM. *(Sanson.)*

Pressure of the bony thorax causes	Local pain. Local œdema (pulsation). Absorption of tissue.	
Pressure on nerve causes	Paralysis	Unequal pupils, paralysis of vocal cords. Hemiplegia or paraplegia.
	Asthmatic dyspnœa.	
Pressure on blood-vessel causes	Inequality of pulses.	
	Obstruction of veins.	Local œdemas. Enlarged collateral veins.
Pressure on air tubes causes	Tracheal signs	Paroxysmal dyspnœa. Brassy cough. Bilateral stridor.
	Bronchial signs	Also paroxysmal dyspnœa and cough. Unilateral stridor. Filling or consolidation of lung behind.
Pressure on lung causes	Consolidation and displacement.	
Pressure on œsophagus causes	Dysphagia.	

Physical Signs. INSPECTION. *1. Ascending Portion of the Arch.*—Bulging of the thorax and pulsating tumor are present to the right of the sternum in the second and third intercostal spaces, except when the aneurism projects upward and inward from the lesser curvature. The apex beat of the heart may be in the normal position or be displaced downward. Pressure upon the superior vena cava causes distension of the superficial veins of the chest and localized cyanosis or œdema of the right side of the face or of the upper extremities. Pressure upon the sympathetic nerve causes contraction of the right pupil. Pressure upon the right or left bronchus causes diminished expansion of the corresponding side of the chest.

2. Transverse Portion of the Aorta.—Bulging of the upper portion of the sternum, with diffuse pulsation most marked in the epigastric notch and in the arteries of the neck. Pulsating tumor is present when erosion of the ribs or sternum occurs. (Figs. 68 and 69.) Trachea displaced backward or laterally. Apex beat is generally in normal position. Pressure upon left bronchus causes restricted motion of the left side. Pressure on the sympathetic nerve causes contraction of the pupil on the affected side.

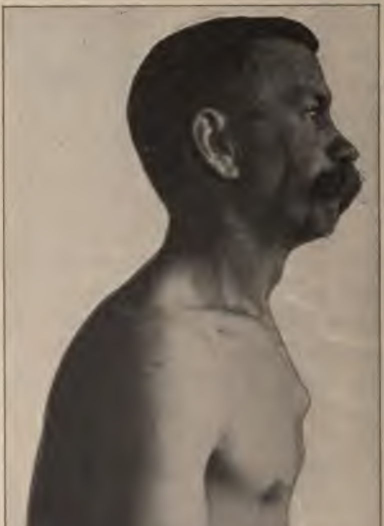
3. Descending Portion of the Arch and Upper Portion of the Descending Aorta.—When the sac develops on the anterior surface, there is bulging of the intercostal spaces to the left of the sternum. The apex beat is displaced to the right, and two areas of pulsation are seen. When it develops on the posterior surface, there is absorption of the ribs and vertebræ, with pulsations in the interscapular space.

4. Descending Aorta, Inferior Position.—Enlargement of the lower portion of the chest, displacement of the heart upward and to the right, diffuse pulsations in the epigastrium and toward the left. Loss of respiratory motion over the lower ribs.

5. Abdominal Aorta.—Pulsating tumor in the abdomen, frequently causing pulsations also over the hepatic area.

PALPATION.—By palpation the dilating or expansile impulse is felt over the aneurism, and associated with a well-marked diastolic shock, due to recoil of the blood upon the aortic valves. In a certain number of cases a well-marked thrill may also be detected. The force of the aneurismal pulsation should nearly equal that of the heart at the apex (Balfour). The location of the apex beat and the occurrence of confirmatory signs will vary according to the site of the tumor.

FIG. 68.



Aneurism of aorta, bulging of sternum and pulsating tumor.

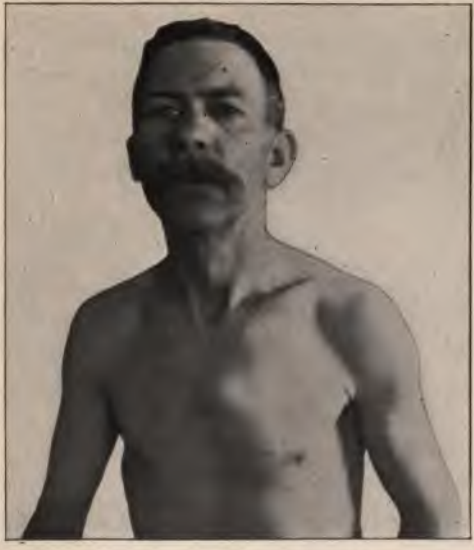
1. *Ascending Portion of the Arch.*—Aneurism of this portion of the aorta is attended by a well-marked

dilating impulse, accompanied with thrill, followed by diastolic shock. The apex beat is displaced downward and to the left, and usually increased in force.

When the aneurism involves the innominate artery, the right radial pulse is usually smaller and slightly delayed.

2. *Transverse Portion of the Arch.*—Over the sternal region the impulse is usually heaving in character, unless absorption and perforation have occurred. Fingers introduced into the episternal notch may detect the dilatation of the aorta, expansile pulsation and thrill. The radial pulse generally shows marked difference in character. Tracheal tugging is almost diagnostic of aneurism of this portion of the aorta. Pressure upon the œsophagus causes dysphagia.

FIG. 69.



Aneurism of aorta, bulging of sternum and pulsating tumor.

3. Descending Portion of the Arch and Upper Portion of the Descending Aorta.—When the aneurism develops forward, the apex beat is displaced toward the right, and may be found to the right of the sternum; while to the left of the sternum the aneurismal pulsation may be detected in the region of the normal apex. The aneurismal pulsation is slightly later than that of the apex. When it develops posteriorly, the apex may be displaced to the right or left, according to the direction of the tumor. Impulse may be felt in the interscapular space.

4. Descending Aorta, Inferior Portion.—The apex is elevated. There is diffuse pulsation over the lower portion of the thorax.

5. Abdominal Aorta.—As the artery can be grasped through the abdominal wall, the dilating character of the pulsation is easily detected.

PERCUSSION.—There is diminished resonance or flatness over the site of the tumor. At times percussion detects the presence of aneurism when inspection and palpation are negative. When the sac projects close to the surface, its size may be definitely determined.

1. Ascending Portion of the Arch.—Aneurism projecting forward and to the right gives well-marked dullness in the second and third interspaces.

2. Transverse Portion of the Arch.—Aneurism pressing upon the sternum causes dullness over the upper portion of the sternum, with increased sense of resistance. When developing posteriorly or backward, increased dullness in the interscapular region may be detected.

3. Descending Portion of the Arch and Upper Portion of the Descending Aorta.—Displacement of the heart causes percussion dullness over an abnormal site. Unless the aneurism projects close to the surface, it may be undetected by percussion. Posteriorly, increase in the percussion dullness may be made out in the left interscapular space from the third to the sixth dorsal vertebræ.

4. Descending Aorta, Inferior Portion.—The normal præcordial flatness or dullness are extended to the left and over the lower portion of the thorax.

5. Abdominal Aorta.—Dullness over site of tumor.

AUSCULTATION.—The signs detected by auscultation are not as distinctive as those noted by inspection, palpation and percussion. Frequently over the aneurism the first and second sounds of the heart are heard with abnormal clearness, and are at times the only signs detected. One or both of the cardiac sounds may be replaced by a murmur or *bruit,* which varies widely in quality. A systolic bruit is most frequently present, and may be made in *(a)* the aneurismal sac, and generally disappears when the contents become solid by coagulation; *(b)* in the dilated artery; *(c)* in a portion of the aorta partially compressed by the tumor. A systolic murmur may be heard over the præcordia (Drummond's sign), or detected when the chest piece of the stethoscope is introduced into the mouth and the lips closed on it (Sanson's sign). Corresponding to the diastolic shock felt on palpation, an intense, low-pitched, second sound may be heard over the aorta. When heard behind in the interscapular space, it is especially significant of dilatation of the aorta. A diastolic murmur is heard when incompetency of the aortic valve is present, and replaces the diastolic shock and sound. The respiratory sounds will be altered according to the site of the aneurism and the pressure on the trachea and bronchi, or displacement of the lung.

1. Ascending Aorta.—Usually, the systolic and diastolic cardiac sounds are either intensified or replaced by murmurs. The most frequent combination is a well-marked systolic *bruit,* and a short, sharp diastolic shock or sound heard over the second intercostal space to the right of the sternum and behind in the interscapular region. Pressure on the right or left bronchus gives rhonchi, tubular breathing (pressure type) and feeble vesicular murmur over the corresponding side of the chest.

2. Transverse Portion of the Arch.—Cardiac sounds and murmurs are similar to those heard when the ascending portion is involved. Pressure on the trachea causes harsh stridor to replace the normal respiratory sounds over both lungs. Pressure limited to the left bronchus gives tubular breathing at point of pressure and feeble vesicular murmur over the left side of the thorax.

3. Descending Portion of the Arch and Upper Portion of Descending Aorta.—Usually distinctive changes in the cardiac sounds and murmurs are not present, and the diagnosis is made from evidences of pressure on the left bronchus and lung and from the displacement of the heart. Narrowing of the left bronchus causes stridor and diminished breathing below the affected portion. Occlusion of the bronchus and collapse of the lung are attended with absence of respiratory murmur and feeble vocal resonance.

4. The Descending Aorta, Inferior Portion.—The auscultatory signs are very slight. Pressure of the tumor does not produce stridor, but may cause over the lower portion of the chest, occupied by compressed lung, bronchial breathing and increased vocal fremitus.

5. Abdominal Aorta.—Usually over the site of the pulsation a well-marked systolic bruit is present. The systolic murmur over the aorta is not pathognomonic of aneurism, as it may occur when the aorta is relaxed, or displaced by anterior curvature of the spine, or slightly narrowed by the pressure of the stethoscope.

Differential Diagnosis.—The distinctive signs of thoracic aneurism are the presence of visible pulsations, with or without bulging of the chest wall, and tumor. Over the tumor, when present, the pulsations are expansile, and frequently accompanied by systolic thrill and diastolic shock. The force of the pulsation felt should nearly equal that felt over the apex beat. The apex beat and area of cardiac dullness may be normal or displaced. Over the site of the aneurism, when it reaches the surface, there may be flatness, surrounded by an area of relative dullness. When deep seated, diminution of resonance only over that portion of the chest may be noted. Auscultation may reveal increased cardiac sounds, with diastolic shock or *bruits* replacing the normal sounds. In addition to the above, pressure signs will be present, varying in character according to the location of the aneurism.

MEDIASTINAL TUMORS.—Mediastinal tumors have many physical signs in common with thoracic aneurism, especially those referable to pressure. The chief distinguishing points are that mediastinal tumors, while producing bulging of the chest, are not attended with erosion of the bony thorax. The pulsations, when present, are not expansile, but convey the impression of a non-expansile force being transmitted. Over the site of pulsation, thrill and diastolic shock are not present. Pressure upon the large interthoracic veins produces more marked superficial venous disturbance.

Glands in the clavicular and interclavicular and axillary regions are involved when the tumor is malignant. Over the site of bulging there is increased firmness of the bony thorax to pressure.

The superficial area of flatness is very much greater than that over which pulsations are felt. Diminution of sternal resonance is especialy marked. Over the site of dullness, increased resistance and loss of normal thoracic elasticity are noticeable.

Over the site of dullness and pulsation, distinctive cardio-vascular sounds are absent. Over the lung, pressure symptoms are more marked and uniform, and apt to be attended by secondary structural changes, due to extension of the growth from the mediastinal.

ARTERIO-SCLEROSIS OR ARTERIO-CAPILLARY FIBROSIS.

This condition causes increased peripheral resistance, with heightened arterial tension.

The effects of arterio-sclerosis can be divided into three stages. First stage—In proportion to the interference with the flow of blood through the capillaries and arteries, there is a corresponding hypertrophy of the left ventricle, which compensates for the lesion as long as the left ventricle is able to empty its contents into the aorta. The second stage or failure of compensation.—When the muscular power of the heart is insufficient to overcome the resistance in the aorta, dilatation of the ventricle occurs, with insufficiency of the mitral valves (mitral regurgitation), and the subsequent changes in the pulmonary circulation, and compensating hypertrophy of the right heart. Subsequently (third stage) failure of the right ventricle to overcome interference with the pulmonary circulation may induce dilatation of the right ventricle, tricuspid regurgitation and interference with venous return.

Physical Signs.—The cardiac changes of arterio-sclerosis are identical with those that occur when the interference is due to obstruction at the aortic orifice. In arterio-sclerosis the obstruction is simply transferred from the area of the aortic valves to a more distant point in the arterial system. (See Aortic Stenosis, page 320.)

The superficial blood-vessels are lengthened, prominent and tortuous, and the pulsations are visible. On palpation the artery is hard and incompressible, and even when the pulsations are obliterated can be felt beyond the point of compression as a hard, rounded cord. On auscultation, the aortic second sound differs from that of aortic stenosis, in that it is accentuated and ringing in quality. With failure of compensation at the left ventricle or regurgitation at the mitral orifice, the aortic second sound becomes indistinct.

PART IV.

THE ABDOMINAL ORGANS.

CHAPTER XV.

INSPECTION.

INSPECTION of the abdomen includes consideration of the shape and size of the abdomen, the changes in the skin, the condition of the blood-vessels, both arterial and venous, and the movements, both respiratory and visceral. The examination should be made with the patient in both the erect and recumbent postures when possible, although usually it is made in the recumbent position only. In the standing posture the shape of the abdomen and the support given the abdominal viscera by the parieties are most clearly seen, and in enteroptosis this method of examination is especially valuable. Examination in the recumbent posture demands that the body shall be exposed from just below the mammary glands as far as the pubes, so as to compare the lower portion of the thorax with the abdomen. The patient should lie perfectly straight on the back, the head and trunk slightly elevated, so as to relax the abdominal muscles. The abdomen should be well illuminated, and the examiner should view it from all sides, so as to note any inequalities of surface and shadows caused by movements.

The Normal Abdomen.—The contour of the abdomen should bear a definite relation to that of the thorax and general physique of the patient. In children and in the adult male past middle life the abdomen is relatively larger and rounder, especially in the upper and middle zones. In males during early adult life there is generally a depression in the epigastric region. In females, independent of the effects of tight lacing, there is more or less of a depression in the lower portion of the thorax and hypochondriac regions, with a lengthening of the waist line and a corresponding broadening of the lower portion of the abdomen. When compression of the thorax by corsets has occurred, this natural condition is made more marked. In those who have borne children, the lower portion of the abdomen is larger and the walls are flaccid.

Examination of the Parietes.—The skin may show certain discolorations (jaundice, pigmentation of Addison's disease and that secondary to pregnancy) or striæ, the result of overdistension (pregnancy, ascites, obesity). It may be tense, dependent upon distension of the abdomen, or lax, secondary to overdistension from pregnancy, ascites, tumors, and in wasting diseases.

The blood-vessels may be more prominent than normal, especially the veins, when the superficial ones are enlarged on account of compensatory changes due to obstruction of return circulation, either portal or general.

The movements of the abdomen are (*a*) *respiratory.* These in the adult male and in children are especially marked. With inspiration the abdomen becomes prominent, especially in the upper zone. In females this is less marked, on account of the breathing being largely costal. With deep respiration the movements are more pronounced in both sexes. Increased respiratory movement occurs in diseases that interfere with expansion of the upper portion of the thorax. Diminished respiratory movement occurs in all painful affections below the diaphragm, either involving the abdominal muscles or the peritoneum, general or local, and especially that portion covering the organs that are influenced by the descent of the diaphragm.

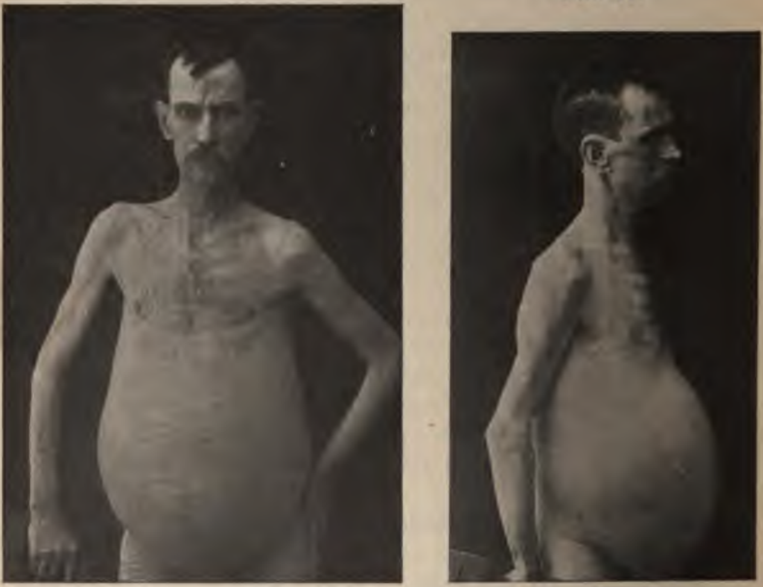
(b) Visceral.—Peristalsis of the normal stomach is not noticed except when the abdominal walls are thin and relaxed. These movements may be noted when the stomach is distended and the peristalsis become exaggerated, especially when there is stenosis at the pylorus, with secondary hypertrophy of the walls.

The movements of the stomach are seen as distinct waves, passing from left to right across the epigastric region to the costal margin on the right side. When more pronounced, they may cause protuberances or bosses of the abdominal walls, which pass slowly in the same direction. The latter conditions occur when the stomach is markedly dilated, and may at times be seen over a large portion of the abdomen.

Movements of the small intestine may normally be seen when the abdominal walls are thin and lax. Those of the large intestine are seen only in pathological conditions, dependent upon neuroses or stenosis, with overdistension of the gut above the site of obstruction. Those of the small intestine are vermicular, and are seen especially in the umbilical region. Those of the large intestine are especialy marked in the lumbar and epigastric regions, and the direction of the motion is contrary to that of the stomach, being from right to left.

Enlargement of the Abdomen.—Enlargement of the abdomen may be (A) symmetrical, (B) localized. Symmetrical enlargement of the abdomen may be due to (1) increase of the abdominal walls (fat, œdema, etc.). (2) Distension of the peritoneal cavity by air (rare) or fluid (ascites). (3) Distension of the intestine with gas (meteorism). (4) Massive tumor filling the abdominal cavity (uterus: pregnancy, fibroids; ovaries: distended bladder, etc. Although these conditions may cause uniform enlargement of the abdomen, certain peculiarities noted by inspection are aids in differential diagnosis. Enlargement due to increase in the abdominal walls is associated with general obesity. The walls in the recumbent posture are more or less widened, and lack the dome-shaped appearance. In ascites the fluid collects in the most dependent portion of the abdomen, which enlarges laterally, and its shape changes as the patient is turned on the side or sits up. (Figs. 70 and 71.) In accumulations of gas in the intestine, the abdomen is distended in all directions uniformly, and there is no change in shape with altered posture of the patient. In tumors the enlargement is not entire symmetrical.

FIG. 70. FIG. 71.



Ascites due to cirrhosis of liver.

Local Abdominal Enlargement or Bulging.—Inspection is very important in detecting local enlargements or bulgings. The abdomen should be viewed from all directions, in order to detect the shadows caused by unevenness of its surface. Local enlargements of the abdomen may be caused by enlargement of structures normally situated in that region or tumors springing from them, or the region may be invaded by organs displaced from adjacent areas.

Epigastric Region. *(1) Abdominal Walls.*—Local contractions of the recti (phantom tumor); abcess of abdominal walls, lipomas.

(2) Peritoneum.—Localized effusions into the lesser peritoneal cavity, tumors of the omentum.

(3.) Stomach.—Distension from food or gas, or dilatation, with or without stenosis; tumors of that portion of the stomach that is parietal (cancer, etc.).

(4) Pancreas.—Cysts, cancer.

(5) Aorta.—Aneurism of the aorta or its branches.

(6) Liver.—Enlargements of the liver, displacement of the left lobe of the liver downwards, abscess, hydatid, gumma, cancer, etc.

(7) Large Intestine.—Distension of transverse colon, tumor of colon.

Umbilical Region.—Many of the conditions that were noted in the epigastrium may extend to this region also.

(1) Abdominal Wall.—Umbilical hernia, abscess, lipoma.

(2) Peritoneum.—Encysted effusion into the peritoneal cavity, tumors of the omentum.

(3) Stomach.—Dilatation, tumors of the pylorus.

(4) Aorta.—Pulsations of aorta due to aneurism or anterior curvature of the spine.

(5) Enlarged Mesenteric or Retro-peritoneal Glands.—Tubercular, cancerous, etc.

(6) Intestine.—Cancer of the intestine, especially the large intestine.

(7) Movable liver; spleen, kidneys may project into this region.

Hypogastric Region.—Distensions may be caused otherwise than by changes in the abdominal wall and peritoneum; they are dependent chiefly upon distended bladder and enlarged uterus (pregnancy, tumors).

Upper Right Lateral Region.—In the lateral region (including the hypochondrium and upper portion of the lumbar region to the level of the umbilicus) enlargements due to changes in the abdominal walls and peritoneum are rare.

(1) Liver.—Enlargements of liver (fatty liver, abscess, congestion, syphilis, hypertrophic cirrhosis, amyloid, displacement of the liver downward, leukæmia, hydatid, carcinoma).

(2) Gall Bladder.—Distended gall bladder, gall stones, hydrops and cancer.

(3) Ascending Colon.—Impaction of fæces, cancer.

(4) Kidney.—Hydronephrosis, pyonephrosis, hæmonephrosis, sarcoma, perinephritic abscess, cancer of suprarenals.

Upper Lert Lateral Region.—*(1) Stomach.*—Dilated, distended, carcinoma of fundus.

(2) Spleen.—Enlargements (congestion, primary or secondary, to obstructive disease of liver; leukæmia, pseudo-leukæmia, malaria, movable or displaced spleen, abscess, carcinoma, hydatid.

(3) Intestine.—Impaction of fæces in splenic flexure of colon. Enlargements of kidney.

Lower Right Lateral Region.—(From the level of the umbilicus to the brim of the pelvis.)

(1) Abdominal Wall.—Hernia, abscess of abdominal wall and psoas abscess burrowing forward.

(2) Peritoneum.—Localized tumor secondary to disease of appendix or intestine.

(3) Intestine.—Tumors of appendix, impacted fæces, typhlitis, perityphlitis, intussception, cancer of cæcum, or of ascending colon.

(4) Kidney.—Displaced kidney.

(5) Ovaries and Tubes.—Ovarian tumors, cysts of

the broad ligament, pelvic abscess, tubal or extra-uterine pregnancy.

Lower Left Lateral Region.—Distension of the sigmoid flexure of the colon due to impaction of fæces, new growths in rectum, in addition to the same conditions may occur on the right side.

Decrease in Size.—The abdomen may be decreased in size, due to contraction of the abdominal muscles, as occurs in the early stage of peritonitis and secondary to cerebral disturbance, especially basilar meningitis. In stenosis of the œsophagus or cardiac portion of the stomach, the empty condition of the stomach and intestines, with the emaciation, causes the abdomen to have a lax, shrunken appearance.

Illumination of the Stomach.—The outline of the stomach may be seen by the use of Einhorn's gastro-diaphane. For this purpose the stomach is first washed out, and then filled with clear water, into which an electric bulb attached to a flexible wire is introduced.

CHAPTER XVI.

PALPATION.

Method of Examination.—For satisfactory palpation of the abdomen, it is necessary for the patient to be in the recumbent posture, and the regions of the abdomen should be accessible from all sides. The head and shoulders should be elevated so as to relax the abdominal muscles, and the patient must be instructed to be as limp as possible, and not try to aid the examination by any voluntary motion. As the object of the examination is to reach the abdominal organs, relaxation of the abdomen is a necessity. Very little is gained by having the patient draw up the legs, as there is a tendency to hold the abdominal muscles rigid in order to support them. When the patient is very nervous or apprehensive of the examination, it may be necessary to divert the attention by asking questions and seemingly to examine the tongue with the eye, while with the hands we are palpating the abdomen. The hands of the examiner should be warm, to avoid causing reflex contraction of the abdominal muscles.

At the beginning of the examination the palms of the hands should be placed lightly upon the abdomen and passed over it in all directions, so as to get a general idea of the condition of the walls, the degree of resistance.

Patients who are very ticklish are frequently more disturbed by a light touch than by more forcible palpation. The amount of pressure should be gradually increased, in order to determine whether any part of the abdomen is sensitive, as shown by pain and local muscular contraction. Whenever such areas are present, their examination should be left till the last.

In general enlargement of the abdomen it is necessary to determine its resistance (gas, fluid or solid). When local enlargements are detected, we determine (1) their relation to the surface, (2) whether they are fixed or movable, the extent and direction of their mobility, and whether they move with respiration; (3) their shape and size, nature of their surface; (4) their relation to structures normally found in that region and to adjacent organs; (5) the presence of frictions, thrills, and pulsations; (6) whether they are sensitive or not.

In order to gain more definite knowledge, it is often necessary after examination in the recumbent posture to put the patient in the lateral or in the knee-chest position.

Abdominal Walls.—The condition of the abdominal walls affects the information obtained by palpation regarding the abdominal viscera.

When the walls are thin and relaxed, they offer very little obstacle to palpation. Tension of the walls, due to muscular contraction, prevents satisfactory palpation, so that an anæsthetic may be required. Localized contractions, especially of the recti, due to ticklishness, pain or tenderness, frequently simulate tumors of the wall or underlying structures.

Increase in thickness of the abdominal walls from fat renders palpation very difficult.

Local enlargements and tumors of the abdominal wall may be due to *(a)* muscular contractions, *(b)* fatty tumors (lipomas), *(c)* abscess, *(d)* hernia. All these tumors give the impression of being superficial. They can be grasped by the hand, and their mobility is limited to that of the walls; while tumors situated within the cavity do not have their mobility limited to the walls unless they adhere to the parietal peritoneum.

Tumors due to muscular contraction vary in shape and size. Contraction of the recti causes a well-defined oblong mass, with the long axis corresponding to that of the muscles, while those of the oblique muscles are ill-defined. The characteristic feature of muscular tumors is their variable consistency; at one time they are hard and resistant, and as the muscle relaxes they become softer, smoother and finally disappear (phantom tumor). They may be painful when due to rheumatism or neuralgia, or attended with irritation of the parietal peritoneum.

Lipomas are soft, yielding masses with fairly well-defined borders, and with greater or less mobility under the skin. Their firmness is increased when the surface over them is chilled by ether spray or the application of salt and ice. They are painless, and when grasped firmly and forced to the surface the skin over them shows depressions, indicative of the lobulated structure of the tumor.

Abscesses are usually tense and elastic or fluctuating; they are well defined unless surrounded by œdematous tissue, which gives a boggy feel and pits on pressure. Acute abscesses are red and painful, with local elevation of temperature.

Hernias. The usual site of the hernial tumor is at the natural openings in the abdominal walls and the umbilicus, although they may occur at any part of the abdominal parietes. The mass gives an air-cushion feel, and becomes more tense as the intra-abdominal pressure is raised by deep breathing, coughing or contraction of the abdominal muscles. They are not usually tender, and firm pressure may reduce the mass with sensation of gurgling. After reduction, the opening in the abdominal wall can be felt. Percussion over the mass gives a tympanitic note.

Separation of the recti muscles leads to projection of the abdominal wall. The tumor is soft, yielding, with air-cushion feel and tympanitic note.

Peritoneum.—Normally, the peritoneum presents no palpable phenomena. Acute general inflammation causes the abdomen to become extremely tender and resistant, on account of muscular contraction. Later paralysis of the intestine causes tympanites to be marked. Respiratory movements are diminished or absent.

General peritonitis may be simulated by painful affections of the abdominal walls (rheumatism, neuralgia, lead colic, etc.) and tympanites, due to intestinal disease, or to hysterical conditions. In painful affections of the abdominal wall, respirations are not disturbed to the same degree as in peritonitis. Pain is more paroxysmal, and the constitutional symptoms are less severe. In hysterical conditions the physical signs may be identical. The diagnosis is made by consideration of all symptoms, especially others of an hysterical nature.

Acute localized peritonitis is usually secondary to disease of organs covered by the serous membrane, and will be considered with each organ.

Diffuse thickening of the peritoneum secondary to acute or tubercular involvement gives an increased sense of resistance, and a more or less boggy feel. In local thickenings and ill-defined indurations there are felt nodular masses, more or less movable, simulating enlarged glands. They are frequently associated with thickening and retraction of the omentum (q. v.).

Fluid in the Peritoneal Cavity, Ascites.—Fluid causes a change in the normal feel of the abdomen, and abnormal sensations. The fluid collects in the most dependent portion of the sac, according to the posture of the patient; and as it increases the intestines are displaced, floating on its surface unless prevented by peritoneal adhesions or short mesentery. Over the fluid the normal air-cushion feel of the abdomen is replaced by increased resistance, as if the wall were thickened. This is especially noticeable in the flanks with the patient in the recumbent posture. Above the fluid the distension of the abdomen causes the walls to be tense, otherwise the feel is normal. With change in posture of the patient, there is corresponding shifting of the fluid and intestines.

Fluctuation.—When the palm of the hand is placed on one side of the abdomen near the level of the fluid, while on the opposite side a sharp percussion tap is made with the other hand, a wave-like motion in the fluid is felt as a distinct impulse, most distinct near the level of the fluid when the fluid is under tension and able to move freely. Under such conditions a light tap is sufficient to produce the phenomenon. When the abdominal wall is thick and the amount of fluid scanty, or if it is prevented from moving freely, the stroke must be stronger, and the impulse is feeble. It is often difficult to determine whether the impulse felt has been transmitted by the fluid or by the abdominal wall. Transmission by the abdominal wall can be arrested if an assistant presses firmly with the ulnar edge of the hand in the median line. When the amount of fluid is small, fluctuation may be best detected by placing the patient on the side and placing a hand over the flank behind, between the lower ribs and the crest of the ilium, and tapping over the front of the abdomen. Fluctuation may be absent when the abdomen is overdistended and tension is very high, or when adhesion of the intestines or a short mesentery prevents the usual motion of the fluid.

The abdomen in ascites is usually painless, unless the condition is secondary to peritonitis or complicated by it. Palpation of solid organs and tumors is prevented when the amount of fluid is large. By *dipping* (*i. e.*, punching sharply with the tips of the fingers) over the organs sought for, the fluid is displaced, and the surface or edge of the solid organs can be felt.

Fluid in the peritoneal cavity may be due *(a)* to inflammation of the peritoneum, simple, tubercular or cancerous; *(b)* interference with portal circulation (obstructive disease of the liver (q. v.), thrombosis of the portal vein, pressure on the portal vein from abdominal tumors, etc.); *(c)* interference with the general return circulation secondary to pulmonary and cardiac disease; *(d)* as a part of general dropsy (kidney disease, anæmic condition).

The differential diagnosis of the cause is made by the physical signs of the primary lesion. Frequently the condition of the liver can only be determined after tapping.

A localized collection of fluid may occur in any portion of the peritoneal sac. It is most common in the lesser peritoneal cavity, where it forms a tense, globular, cyst-like tumor in the epigastric region, not movable, and over which distinct fluctuation cannot be obtained. It is generally painless. At times it may transmit from the aorta pulsations which are not expansile.

Air in the peritoneal cavity, due to perforative peritonitis, gives marked distension of the abdomen, with increased tension of its walls. The diagnosis from tympanites is made by percussion and auscultation.

The Omentum is not palpable unless thickened by inflammation or new growths. Deposits of fat in the omentum (peritoneal lipoma) give a thickened, elastic feel to the abdomen, with the impression of fluctuation. Thickening of the omentum may occur from chronic or tubercular peritonitis, and gives the sensation of increased superficial resistance over the upper zone of the abdomen, without any free border being detected. There may be tenderness in the acute and subacute stages, when more chronic there is no pain. The mass is *not* movable with respiration. Retraction of the thickened omentum may form a distinct tumor in the upper portion of the abdomen. The tumor may be felt as a transverse ridge with a distinct border below, which may simulate an enlarged left lobe of the liver, or it may be an irregular or ball-like mass. Cancer nodules may be diffused over the omentum, or new growths may form irregular masses similar to those in the stomach and liver. Tumors of the omentum do not move with respiration unless attached to the edge of the liver, in which case differential diagnosis is very difficult. They are differentiated from primary malignant growths of the stomach by being fixed, and more prominent when the stomach is full or inflated.

Hydatid cysts of the omentum are rare. When unilocular, they give a sensation of superficial, globular tumors, and the hydatid thrill may be present. Multilocular cysts, when small, may simulate the early stage of cancerous infiltration.

Stomach.—Normally, the stomach cannot be detected by palpation, as only a small portion of the anterior surface and greater curvature is in contact with the abdominal wall. In extreme emaciation, during contraction of the stomach, there is slight resistance. Normally, in most persons the upper portion of the epigastrium is more sensitive to pressure than the rest of the abdomen. Increased tenderness over the gastric region occurs in acute and chronic catarrhal inflammation of the stomach; in malignant disease, in ulcer (circumscribed area) and whenever the peritoneal covering of the stomach is involved secondary to gastric disease.

In dilated or displaced stomach the boundaries cannot be determined by palpation. The swelling noted by inspection gives a soft, yielding feel, except during contraction, when the sensation varies according to the cause of the dilatation. In atonic dilatation there is slight increase of resistance, and an even, wave-like motion from left to right can be detected. When dilatation is due to obstruction at the pyloris (cancer, ulcer, stenosis from adhesion, or displacement) there is hypertrophy of the muscular coat, and the contractions of the stomach causes hard masses and irregular protuberances to be felt as the peristaltic wave passes across the abdomen. Over the region of the pyloris the contraction is harder and longer, and almost always accompanied with sensations of gurgling or escape of gas. When the stomach relaxes a tumor may be felt.

It is only exceptionally that the normal pylorus can be felt as a tumor. Splashing and succussion sounds (clapotage) can be obtained in a dilated stomach when it contains both gas and fluid. They are obtained by placing the tips of the fingers over the stomach and shaking it with quick movements, or by bimanual palpation, one hand beneath the ribs behind and the other over the stomach in front. They are diagnostic of dilatation only when taken in connection with other signs, as they can be obtained in the normal stomach, and in the colon when the contents are liquid.

TUMORS OF THE STOMACH.—Tumors of the stomach can only be felt when situated in that portion which is in contact, normally or abnormally, with the abdominal walls. They may be due (1) to contracted stomach, (2) diffuse infiltration of the walls, with cancer; (3) local thickenings of different portions of the stomach wall by inflammatory products or new growths.

The entire stomach may be so contracted as to form a tumor *(a)* in stenosis of the œsophagus or cardia, *(b)* by the prolonged use of concentrated diet, *(c)* by fibrous thickening of the stomach, with or without cancerous infiltration. In these conditions the stomach is usually a rigid mass, felt as a transverse ridge in the region of the normal stomach, and giving the same sensation as a thickened omentum. It is movable, and changes its position with deep breathing.

Diffuse infiltration of the stomach wall may be later associated with contraction of the stomach and formation of a tumor, as mentioned above, or the lumen of the viscus may be increased. In the latter condition the stomach is felt as a large, ill-defined mass, which, during contraction, becomes harder and has the more definite contour of the stomach. It may occupy the normal gastric position, but is usually displaced downward, and the pylorus is felt somewhat to the left of the median line.

TUMORS OF THE PYLORIC REGION. These may be due (1) to primary changes at the pylorus, as already mentioned, or to extension of cancerous growths from neighboring organs to this region.

The characteristic features of primary tumor of the pylorus are *(a)* their location; usually they are detected to the right of the median line, between the umbilicus and the ensiform cartilage. When stenosis is marked, dilatation and displacement of the stomach cause the tumor to be felt in the region of or below the umbilicus and to the left of the median line. *(b)* Mobility. When felt in the epigastric region, the tumor is movable in all directions, but more freely downward and to the left. When the pylorus is displaced below the umbilicus, the mobility is not so great. *(c)* The tumors vary in size, and are usually actually larger than palpation would indicate. *(d)* Their consistency is hard; during peristalsis they become harder, and their contour more defined. *(e)* The detection of gurgling or escape of gas is an important diagnostic sign, especially when it occurs during peristalsis.

Tumors of the pylorus, due to extension from neighboring organs, lack the characteristic mobility, and are usually associated with the physical signs of the primary growth.

Pancreas.—The normal pancreas is not palpable, although claim has been made that in extremely thin persons and in those with enteroptosis it can be distinctly felt. In diseased conditions the gland can only be detected when it is enlarged to a considerable degree. In acute or chronic pancreatitis it is not palpable, but in the epigastric region tenderness on firm pressure may be present with increased sense of resistance.

Pancreatic enlargements are mostly due to cancer and cysts. Tumors due to tubercle, gumma, lymphoma and sarcoma are rare, and when present give most of the physical signs of cancer.

CANCER OF THE PANCREAS.—As this usually begins at the head of the gland, a tumor is felt in the epigastrium, midway between the xiphoid cartilage and the umbilicus, and a little to the right of the median line, close to the edge of the liver. Its form may be round or oblong, but the outline cannot be well defined. Its position is not influenced by respiratory movements. When it can be easily felt, it is generally fixed, or only slightly movable. Non-expansile pulsations may be felt, due to its relation to the aorta. Compression by the tumor of the portal vein causes ascites; that of the vena cava causes œdema of the lower extremities. The presence of a tumor in the region of the pancreas is not sufficient for diagnosis of cancer of that organ, as similar tumors may be caused by cancer of the pylorus, duodenum, colon, or liver. The associated signs of cancer of the pancreas are (1) early, intense and persistent jaundice; (2) dilatation of the gall bladder, (3) enlargement of the liver, (4) fatty stools.

Differential Diagnosis.—It is differentiated from tumor of the pylorus by being deeper seated and not movable, and that the stomach is not dilated and hypertrophied. When the growth extends to the duodenum, stomach changes may be present. Tumors due to cancer of the lower edge of the liver, in the region of the pancreas, move with respiration, and their relation to the edge of the liver can usually be determined. In primary cancer of the gall bladder the distended gall bladder is harder, while the liver is not enlarged. Enlargement of the retroperitoneal glands from any cause may give a tumor similar, as far as palpation is concerned, to cancer of the pancreas.

Cysts of the Pancreas.—When small, the tumor is felt to the left of the median line, toward the costal cartilage. It is globular, smooth, resistant and inelastic, and when deep seated gives a sensation of hardness. It is uninfluenced by respiratory movements, and on firm pressure may be slightly movable, both vertically and laterally. As the cyst enlarges, it is more in the median line, and displaces the stomach upward (rarely down-

Differential diagnosis between displacement of the liver without enlargement and true enlargement of the viscus is made by the percussion outline.

(B) I. UNIFORM ENLARGEMENTS of the liver may be due (1) to circulatory changes which may be active, as occur in hot climates and in acute inflammatory conditions, and those dependent upon infectious diseases (malaria, typhoid and scarlet fevers, measles, yellow fever, small-pox, etc.); passive congestion dependent upon interference with circulation in the hepatic vein, due to cardiac or obstructive pulmonary diseases or pressure on the vena cava. (2) Blood diseases. Leukæmia, Hodgkin's disease, and pernicious anæmia. (3) Degenerations of the liver (fatty liver, amyloid liver, hypertrophic cirrhosis, diffuse syphilitic hepatitis.). (4) Obstructive disease of the bile ducts (jaundice), either primary or secondary.

II. IRREGULAR ENLARGEMENTS OR TUMORS OF THE LIVER, due to cancer, hydatid and abcess, also syphilitic liver. These conditions may produce uniform enlargements. At times irregular enlargement of the liver may be due to prolongation of the lower portion of the organ and from the effects of pressure of tight lacing.

(1) Congestions of the liver. In simple, active hyperæmia of the liver the enlargement is slight. The lower border is felt just below the free edge of the ribs and slightly lower than normal in the epigastric region. The consistency is slightly increased, the surface is smooth and the edge well defined. In acute infectious diseases, especially typhoid fever in the early stage, the enlargement is identical with that of simple congestion, later changes in the liver cause it to become larger, softer, and the lower margin cannot be so readily felt. In other infectious diseases the liver shows varying degrees of enlargement and consistency, but the normal shape remains. Tenderness is present whenever the peritoneal covering is stretched or inflamed.

In passive congestion of the liver the enlargement is in proportion to the amount of interference with the circulation. Secondary changes cause the organ to become harder; the surface is smooth, the edges sharp and well defined. With secondary interference with the portal circulation, ascites occur. Jaundice occurs in secondary intestinal catarrh. Chronic congestion of the liver may also be due to malarial infection and residence in the tropics.

(2) In leukæmia, Hodgkin's disease, pernicious anæmia and diabetes the enlargement of the liver is uniform, and varies with the severity of the disease. In leukæmia and Hodgkin's disease it is relatively less marked than the enlargement of the spleen and lymphatic glands. The normal outline of the organ is retained; it is harder than normal, and the edges are usually sharp and well defined; the surface is smooth.

(3) In fatty liver the organ is uniformly enlarged and of normal shape. The enlargement may be very marked, and the lower edge reach almost to the crest of the ilium and below the umbilicus. The feel is soft and elastic; the surface is smooth, the edges round and well defined, and the outline can be easily made out by slight pressure. It is not tender to pressure, and is not attended with interference with portal circulation or enlarged spleen.

Hypertrophic cirrhosis has many of the features of fatty liver. Its enlargement may be great, and it is harder than normal; the surface is smooth, and the edges sharper and well defined. With contraction of the connective tissue, there is diminution in size; the liver becomes harder, the surface and edges irregular, but the nodules are small, assuming gradually the features of cirrhosis with enlargement. The organ is not tender to pressure unless associated with perihepatitis.

In amyloid liver the enlargement is marked, but the liver has a hard, resistant feel, and the surface is smooth, with sharp, well-defined edges; sometimes, however, they may be rounded. The edge of the liver cannot be moulded by firm pressure. Pain and tenderness are absent. Ascites occurs in the later stages.

In diffuse syphilitic hepatitis the liver is larger and firmer than normal; the surface is smooth; the edges are sharp and well defined, similar in many respects to the condition noted in amyloid liver.

Syphilis of the liver may cause the organ to be nodular when gummata are formed, and when contraction of the fibrous tissue causes depressed areas with intervening nodules or bosses of hypertrophied liver tissue. The enlargement may be marked, extending nearly to the umbilicus. The consistency of the nodular masses varies according to the amount of connective tissue present. Gummata, when close to the surface, are more resistant than normal liver tissue. The enlargement of the liver is not rapid, and the nodules increase in size slowly.

Cancer of the liver may cause enlargement to a varying degree. Though it is usually massive, it may involve certain portions of the organ to a greater extent than others; but the enlargements may be fairly uniform. The consistency is not uniform, the cancerous masses being harder than other portions. The surface is irregular, and the irregularities may be felt as bosses or nodules of varying sizes. Frequently at the apex of a nodule a depression (umbilication) can be felt, and this is diagnostic of malignancy. These tumors may, however, be soft or semi-fluctuating. The edges are usually irregular, due to the projection of cancerous growths and to contraction of the liver substance. There is more or less tenderness on pressure over the prominent areas when the new growth involves the peritoneal coat. Ascites and jaundice occur in 50 per cent. of all cases.

Abscess may develop in different portions of the liver, and cause irregular enlargement upward into the thorax or downward below the free border of the ribs and in the epigastric region, or outward through the thoracic walls. When central, it may cause a more or less uniform enlargement of the organ. When it develops upward, the liver is displaced downward, but has the normal outline and characteristics. When the tumor develops from the lower portion of the liver, it is more or less globular; its surface is smooth, and fluctuations may be detected when the abscess is close to the surface. Pain and tenderness are variable.

Hydatid cysts of the liver may cause enlargement similar in many respects to that of abscess. When the cyst is situated on the lower border of the organ, the mass is globular, soft and fluctuating, and at times the hydatid thrill may be detected. They are painless unless attended with localized peritonitis.

(4) Obstructive disease of the bile ducts, primary and secondary, causes hepatic enlargement. The enlargement is usually slight, the consistency but little firmer than normal, the surface smooth and the edges well defined. Interference with portal circulation and enlargement of the spleen occur according to the nature of the cause (see Gall Bladder).

Differential Diagnosis.—Differentiation by palpation of the uniform enlargements of the liver from each other and from displacements through thoracic or subdiaphragmatic conditions has been described in considering the different diseased conditions of the organs, and will be further discussed under percussion and auscultation.

The irregular enlargements may be simulated by other tumors occurring in organs adjacent to the liver. The most prominent of these are accumulations of fæces about the hepatic flexure of the colon (see Intestine), cancer of stomach (see Stomach), right renal enlargement (see Kidney), puckered or indurated omentum (see Omentum), and tumors of the abdominal wall. Usually the differentiation is made not only by the signs obtained from palpation, but from those obtained by percussion and auscultation, together with the secondary effects of these tumors upon organs with which they are connected.

The most important features of tumors of the liver are their mobility, their parietal situation and their well-defined relation to the edges of the liver, jaundice and ascites. Cancerous growths that involve the liver and neighboring organs late in the disease have no distinctive features by which the origin of the growth can be determined, and the differential diagnosis is made from the history and the secondary effects.

Gall Bladder.—When it is empty, the gall bladder cannot be felt, but when moderately distended it may be detected as an ill-defined mass at the edge of the right ninth costal cartilage, close to the outer edge of the rectus muscle, which corresponds to that point at the free border of the ribs where a line drawn from the right acromian process to the umbilicus crosses it.

Palpable enlargements of the gall bladder are due (1) to distension by *(a)* bile, *(b)* mucus (hydrops), *(c)* pus (empyemia) and *(d)* to gall stones which may be caused by inflammatory conditions or stenosis or obstruction in the cystic or common ducts. (2) To new growths, malignant and benign.

Enlargements of the gall bladder, from whatever cause, have certain features in common. The size varies greatly from a scarcely palpable tumor to one of large size; usually, however, it is not larger than a good-sized pear. The degree of enlargement of the same tumor may vary from time to time. The tumor is smooth, rounded and gourd-like, the large end being the most dependent. When the tumor becomes very large, the lower portion becomes more globular in relation to the neck, which is thin. When the distension is due to gall stones, the tumor may have a nodular feel.

The tumor has a double mobility, moving with the liver with respiration, and also being more or less freely movable in all directions about its point of attachment to the liver, the range of motion being one of the

E LIVER.—LEUBE.

Dim	Thick, ounded.	Nodular.	Smooth.	Nodular.
	EDGE OF LIVER.		SURFACE OF LIVER.	
Simple Atroph liver. Atroph Syphili (atro rare). Acute y phy.	ty liver. gestion. yloid.	Cirrhosis (rarely palpable). Abscess. Carcinoma. Syphilitic liver. Tubercle bacilli.	Congestion. Fatty liver. Jaundice, obstructive. Elephantiasis. Amyloid. Leukæmia. Diabetes liver. Acute yellow atrophy.	Cirrhosis. Abscess. Syphilitic liver. Carcinoma. Echinococcus.

ENLARGEMENT OF THE SPLEEN.

A	bsent.	Present.
Amylo Fatty l Adhesi phle	inoma. y liver.	Echinococcus unilocularis (rarely from stasis in the portal system). Congestion. Syphilitic liver. Cirrhosis (atrophic). Echinococcus multilocularis. Hypertrophic cirrhosis. Amyloid. (Also in acute yellow atrophy and abscess due to the general systemic infection.)

N ase *increases in frequency* or intensity as the column is read

diagnostic features of the tumor. Generally it can be carried up and caused to disappear beneath the liver, and can also be carried to the median line; but it is impossible to displace it downward toward the pelvis.

Its relations to the liver are well defined; a sulcus can be usually detected between the enlarged gall bladder and the liver. Fluctuation cannot be detected, as the tumor is usually too tense. The tenderness varies. When due to obstruction from gall stones, pressure over the tumor may cause colicky pains.

Malignant growths of the gall bladder are usually hard and more or less irregular. The size in this condition is not so great as in distension by its contents.

Enlargement of the gall bladder may be associated with enlarged liver when obstruction of the common bile duct occurs from any cause, and jaundice will then be a marked and persistent symptom. When the obstruction occurs in the cystic duct, jaundice, if present, is transient, and not intense.

Differential Diagnosis.—Tumors of the gall bladder may be confounded with prolongations of the lower edge of the liver, with hydatids of the liver, with movable kidneys, tumors of the intestine and tumor of the pylorus of the stomach.

(1) Abnormal prolongations of the lower border of he liver do not have free mobility, and their outline is sually continuous with the smooth, convex surface of e liver. Their size is permanent.

(2) Hydatid tumor attached by a pedicle may simute an elongated gall bladder. It is more distinctly ctuating, and frequently hydatid thrill can be tected. It is painless, and, while it is movable, has the same range of mobility as has the gall bladder, l it is displaced more slowly.

3) Movable or floating kidney has many features in mon with enlarged gall bladder. Both tumors are ded and smooth, but that of the gall bladder is d-shaped, the narrow portion projecting toward the

fissure of the liver, the enlarged lower end lying toward a point just below the umbilicus. The kidney retains its normal shape. The feel of the two is also somewhat different. The gall bladder is usually firmer, and when filled with calculi may show an irregular outline. The gall bladder gives the impression of being superficial, and it is constantly felt when pressure is made over the front of the abdomen. Movable kidney, on the other hand, is variable in its situation, and it is not always detected in the same position by anterior palpation. The two tumors differ in range of mobility. The gall bladder is influenced by respiration, and the kidney, when situated close to organs that move synchronously with the diaphragm, may also have a similar motion. The gall bladder moves around a fixed point, and can be pushed upward to either side and backward, but not downward into the pelvis; and when displaced behind the liver towards the normal kidney-position it tends to return to its own normal position in front of the abdomen. The kidney moves readily to different locations in the abdomen. It may be carried to the median line and beyond or downward into the pelvis, and upward and backward into the normal position, where it tends to remain until displaced by pressure over the loin or by posture. It slips underneath the examining fingers like a "greasy mass."

When the large intestine is inflated, the gall bladder is pushed up and becomes more prominent. The movable kidney disappears behind the intestine, and cannot be felt.

Tumors of the pyloric region of the stomach, while movable, do not have the same range of motion, being displaced farther to the left beyond the median line, with a more restricted mobility to the right. Associated conditions are usually sufficient for diagnosis.

Spleen.—The spleen is deeply situated underneath the bony thorax posteriorly, and is normally not palpable, except when the abdominal walls are thin and lax and the spleen is slightly movable, and can be pushed forward so as to be brought close to the costal margin in the hypochondrium. Frequently, when the spleen is abnormally movable, turning the patient on the right side causes it to have an anterior position, and to be felt as a movable tumor. Deformity of the thorax, especially when involving the spine, may cause the normal spleen to be palpable.

Movable or wandering spleen gives, in addition to the tumor that is felt at the free margin of the ribs, a diminished sense of resistance on palpation over the normal area, and a more tympanitic percussion note.

ENLARGEMENTS OF THE SPLEEN.—Conditions that increase the spleen in size cause it to develop anteriorly, and to be felt under the free border of the ribs. The enlargements may be uniform or irregular.

Uniform enlargement occurs *(a)* in infectious and febrile diseases. In many of these the enlargement is so slight as to be scarcely palpable. In typhoid fever, scarlet fever, small-pox, it is generally palpable, according to the severity of the disease. In erysipelas, sepsis and pyæmia, also in acute tuberculosis, the enlargement of the spleen may be sufficient to bring its free edge beyond the border of the ribs. *(b)* In interference with the portal circulation from primary disease of the liver (q. v.), or to cardiac or respiratory disease. *(c)* In chronic hypertrophy, due to amyloid disease, leukæmia, Hodgkin's disease, splenic anæmia, chronic malarial infection (ague cake), and occasionally in syphilis and tuberculosis.

The irregular enlargements occur chiefly in hydatids, cancer and abscess.

The characteristics of splenic tumor are: (1) They are superficial, and are not separated from the abdominal walls by any of the abdominal contents. The upper portion disappears under the free border of the ribs, and cannot be defined. (2) They move with respiration, and have also a slight independent range of motion upward and backward on firm pressure. They have passive mobility toward the median line and downward when the patient is turned on the right side and put in the knee-chest position. (3) The tumor in uniform enlargements retains the normal splenic contour, and when it extends any distance beyond the free border of the ribs the notch can be easily detected. (4) The surface of the tumor is smooth. (5) The consistency varies. In acute enlargements, due to infectious and septic diseases, it is soft and yielding; in the more chronic enlargements it is hard and resistant. In the irregular enlargements of cancer, hard, nodular bosses may be felt on the surface. In hydatid cyst, when the tumor can be readily palpated, it is tense, elastic and rounded; fluctuation, however, can rarely be detected, but hydatid thrill may be obtained.

(6) The edge of the spleen is usually well defined, and, with the notch, is one of the diagnostic features.

When the consistency of the spleen is soft, the edge is not so distinct as in some chronic enlargements, especially malarial.

The size of the spleen varies in different enlargements. In the chronic enlargements of leukæmia, Hodgkin's disease and malaria the size is greatest. The spleen is rarely painful, except when the peritoneal covering is involved, as occurs in acute inflammation, infarction, syphilis and abcess. Posteriorly, the splenic enlargement does not extend to the median line, so that forcible palpation below the ribs in the flank may enable us to distinguish the edge between the edge of the tumor and the erector spinæ muscles.

Differential Diagnosis.—The acute enlargements of the spleen cannot be differentiated by palpation. When dependent upon engorgement, due to obstructed portal circulation, the enlargement is usually associated with changes in the liver and heart, and there is more or less ascites.

Of the chronic enlargements, amyloid enlargement is rarely great. It is usually associated with similar changes in the liver and kidney, due to prolonged suppuration. In leukæmia the splenic enlargement is marked, and is attended with anæmia and changes in the superficial glands and liver. In Hodgkin's disease the splenic enlargement is relatively marked, as is also the enlargement of the lymph glands. In ague cake the enlargement closely resembles in size and consistency that of splenic leukæmia, and the differential diagnosis is made on the blood examination.

Enlargement of the spleen in anæmia (splenic anæmia) occurs chiefly in children, and is not attended with blood changes of leukæmia or malarial infection.

Splenic enlargement may be confounded with other tumors appearing in the left upper portion of the abdomen, the most important of which are fecal accumulations in the descending colon and sigmoid flexure. Fæcal masses are distinguished by the absence of respiratory and passive motion, the rounded contour, absence of the notch and sharp edge, the peculiar feel, and the effect of treatment (purgatives).

Cancer of the stomach, especially of the greater curvature, and the cardia may give an ill-defined tumor under the free border of the ribs. When the tumor becomes palpable in the epigastrium or below the ribs, the hard feel, associated with gastric symptoms, readily distinguish it.

The differential diagnosis between tumors of the left kidney and spleen will be considered below.

Kidneys.—The normal kidney cannot be detected by palpation, except in children and in persons who are thin and have very lax abdominal walls, when the lower edge can be felt on bimanual palpation just underneath the free border of the ribs, at the end of a deep inspiration. The kidneys may be palpable when they are abnormally movable or wandering, or enlarged.

Movable kidneys are those which have a range of motion downward toward the pelvis, and which do not extend laterally into other portions of the abdomen. In this condition, when the patient is standing and leaning forward, or at the end of deep inspiration when in the semi-recumbent posture, the kidney may be felt to glide under the hand into the pelvis, so that the upper border can be felt. On change of position or pressure from below, the kidney slips back into its normal position. The cause of movable kidney may be enteroptosis or emaciation in a person who has once been very stout. It is especially liable to occur in women who have borne children and with overstretched abdominal walls. Anterior curvature of the spine, involving the lower dorsal and lumbar vertebræ, frequently displaces the kidney forward, so that it is both seen and felt in the anterior portion of the abdomen.

Wandering kidneys have a wider excursion, and may occupy any part of the abdomen, moving from place to place with ease, according to the posture of the patient and the direction of pressure. The kidney shape is retained, which enables one to recognize the organ; the borders are rounded, and the hilus is frequently palpable. On firm pressure pain of a sickening character is elicited, frequently radiating down toward the bladder.

ENLARGEMENTS OF THE KIDNEY.—Enlargements of the kidney are due to (1) malignant disease (sarcoma and carcinoma), (2) cystic degeneration, (3) hydatid, (4) hydro- and pyo-nephrosis, and (5) perinephritic abscess.

Renal tumors are extremely difficult to diagnose, and are frequently confounded with those of other organs. Their most characteristic feature is their relation to the surface. When they reach the anterior portion of the abdomen, their most prominent part is at the umbilicus or just above it. They occupy the space between the crest of the ilium and the costal margin. Posteriorly, they fill the entire space between the lower border of the ribs and the pelvis, causing a smoothing out of the hollow of the loin, but rarely any prominence.

Forcible palpation in this area gives a uniform resistance, extending to the spine. The mobility of the tumors is but slightly influenced by respiration, although when they are large and in contact with the diaphragm, a slight respiratory mobility may be detected, but it is never as marked as in tumors of the liver and spleen. The statements that kidney tumors are totally uninfluenced by respiration frequently lead to error in diagnosis.

The size of the tumors varies greatly, and also that of the same tumor from time to time. Tumors due to sarcoma, cystic degeneration and perinephritic abscess are stable in size, and may reach great dimensions, especially sarcoma in young subjects, almost filling the entire abdominal cavity. They rarely cause projection of the lower ribs or fill the pelvis. Those due to hydro- and pyo-nephrosis are more variable in size, according to the degree of distension.

The uniform enlargements preserve to a marked degree the kidney shape. Perinephritic abscesses, on the other hand, may be somewhat globular.

The consistency of the tumor varies, being hard and dense in malignant disease and elastic in other forms, varying with the degree of tension.

Fluctuation can rarely be detected. The relation of enlargement of the kidney to other structures is somewhat characteristic. The large intestine usually overlies the tumor, and can be detected by palpation and percussion. (Fig. 74.) The small intestine may also be between it and the abdominal wall. In exceptional cases the tumor may displace the colon downward and toward the median line on the right side, or carry the descending portion of the colon toward the median line so as not to be covered with it. This relation of the large and small intestines to the tumor overlying it distinguishes it from splenic tumors (Fig. 73), which are always parietal and never have the gut between them and the abdominal wall, and, on the other hand, liver tumor, which is usually parietal, but may occasionally have the small intestine between the abdominal wall and itself when the enlargement is irregular and is associated with ascites.

IRREGULAR ENLARGEMENTS.—Irregular enlargement of the kidney by malignant disease or abscess may cause the tumor to lose its reniform shape, making it abnormally prominent in one direction; and may also change the normal relation to the intestine.

Differential Diagnosis.—Renal tumors are differentiated from splenic tumors by the rounded contour and the absence of the sharp, well-defined edge and notch; also by the presence of intestine over the tumor (percussion). The renal tumors are less movable. The renal tumors, if they passed beneath the ribs, leave a palpable sulcus, due to their rounded border. Splenic tumors are in close contact with the anterior surface. Posteriorly, renal tumors occupy the entire space between the free border of ribs and crest of ilium, and the resistance is uniform. In splenic tumors an unoccupied area can be detected in the flank, which is also resonant on percussion.

Hepatic tumors are also parietal at the costal margin, and the free edge of the tumor, with the sharp, well-defined margin, is transverse to the abdomen, following the normal slant of the liver. When renal tumors develop upward, so as to be in contact with the liver, a sulcus can usually be detected between the rounded edge of the tumor and the sharp, displaced edge of the liver.

Ovarian tumors are parietal, and are surrounded with intestines, giving resonance on percussion. They grow upward from the pelvis, and reach the surface just below the umbilicus, while renal tumors generally develop from behind forward, and first reach the abdominal wall at or a little above the umbilicus.

The massive ovarian tumors generally become centrally situated, while renal tumors remain unilateral. Ovarian tumors cause displacement of the uterus upward or downward. Renal tumors rarely involve the pelvis.

Intestines.—The different portions of the intestine cannot be distinguished from each other by palpation. The small intestine normally is not palpable. It is possible to feel certain portions of the large intestine when the abdominal walls are thin and relaxed. The sigmoid flexure of the colon can most frequently be detected, and occasionally the head of the cæcum and ascending colon. The transverse portion cannot be made out. The appendix is only at times palpable, although the statement is frequently made that it is possible to feel it both by surface and bimanual palpation, one finger being in the rectum or vagina. Different portions of the intestinal canal may become palpable when distended with gas or the walls become thickened through muscular contraction or inflammatory products or new growths, or when the canal becomes filled with semi-fluid or solid contents. In simple distension of the intestines, with relaxation of the intestinal walls, the entire abdomen has an air-cushion feel. When the distension is the result of chronic obstruction in any portion of the tract, the hypertrophied intestine may be more or less distinctly felt during peristalsis.

Localized inflammatory thickening of the intestinal wall may occur at any point. It is rarely palpable in the small intestine, as it is usually associated with narrowing of the part and distension of the intestine immediately adjacent.

Thickening of the cæcum, appendix and ascending colon causes a tumor to be felt in the right iliac region. The appendix may be detected as a hard, cord-like mass, more or less freely movable under the finger. When acutely inflamed and associated with peritoneal involvement and rigidity of the muscles, the appendix cannot be distinguished as such, but an ill-defined tumefaction or a distinct tumor occupies the appendicular region, with a marked tenderness at a point midway between the anterior superior spine and the umbilicus (McBurney's point).

Involvement of the cæcum, with extension of inflammation to surrounding parts (typhlitis), causes a similar tumor to be felt. Abscess formation (perityphlitic abscess) in this region, due to inflammation of either appendix or cæcum, gives a less resistant tumor, with a sensation of deep-seated fluctuation.

Cancer of the head of the cæcum and iliocæcal valve, producing stenosis, causes a tumor that is hard, resistant, and, unless the neoplasm has extended to surrounding structures, nodular and movable.

The tumor of intussusception in this region is smooth, uniform, and not tender on pressure. It is also associated with distension of the intestine, and may not be palpable. Tympanites is marked.

Fæcal impactions, occurring in the ascending colon, give an oblong tumor with a rounded contour, which may extend from the middle of Poupart's ligament to the under surface of the liver. The surface may be smooth, but it is usually more or less irregular or lumpy, and on firm pressure an indentation may be caused. When the pressure is removed, the walls separate slowly from the mass, giving a sensation of stickiness (adhesive sympton). When the fæcal mass is hard, these symptoms may be absent, and the feel may closely simulate that of a cancerous tumor.

Next to the appendicular region, the most frequent site of tumors is the left iliac region. The most common tumors here are inflammatory thickening of the intestine secondary to diseases causing ulceration. Inflammatory thickening of the sigmoid flexure frequently causes it to be felt as a hard, well-defined, rope-like mass. Fæcal impaction and malignant disease in this region have the same features as elsewhere.

In examining the intestinal tract by palpation, in addition to tumors, sensitive and painful areas must be noted. The different portions of the canal vary greatly in their sensitiveness to palpation. It is most sensitive in the region of the cæcum, and epigastrium midway between the ensiform cartilage and umbilicus. The entire canal may become sensitive in acute intestinal irritation, whether inflammatory or not. Local inflammation of the intestine causes increase in sensitiveness, and when the peritoneum is involved there is localized pain, with increased rigidity of the overlying muscles.

Differential Diagnosis.—In addition to the tumors already noted, the ilac regions may be invaded by tumors from the pelvic organs, as ovarian tumors, cysts, especially of the broad ligaments, pelvic abscess, extra-uterine pregnancy, or by hernias and abscess burrowing along the psoas muscles. These are differentiated usually by vaginal examination.

Pelvic Organs.—Tumors may arise from the uterus and adnexa. Tumors of the uterus may be due to pregnancy, fibro-myomata or fibroids. In pregnancy the tumor is ovoid, smooth, freely movable laterally, and in proportion as it extends above the pelvic brim the diagnostic signs of contractions and fœtal movements are detected. Distension of the uterus from retained menstrual flow and from growths may give the contractile symptoms of pregnancy.

Fibroid tumors of the uterus may simulate pregnancy, as far as size, shape and mobility are concerned, but they lack the contractile sign. When they involve only one portion of the uterine wall they give an irregular tumor.

Ovarian tumors are usually felt in the iliac regions, and as they increase in size assume more the medial position. They are usually cystic, and have a round, smooth, elastic feel. Fluctuation can at times be detected. Examination per vaginam shows their relation to the broad ligaments and uterus. They are differentiated from ascites and renal tumors by percussion.

Parovarian cysts have many features of ovarian cysts. As they are usually thin-walled, fluctuation is a marked symptom.

Distended bladder may give a tumor occupying the lower portion or the entire abdominal cavity. Its shape, smoothness and elasticity cause it to simulate ovarian tumors, ascites, pregnancy and pancreatic cysts. The possibility that a large abdominal tumor occupying the median position may be a distended bladder should always be borne in mind, and the viscus should be examined by catheter before excluding this condition.

CHAPTER XVII.

PERCUSSION.

By percussion it is possible to outline the borders of the solid organs of the abdomen, and by the relative degree of dullness or flatness to determine their relation to the air-containing (resonant) organs of the thoracic and abdominal cavities.

When two solid organs are in contact, as the left lobe of the liver and heart, it is impossible to define their borders by simple percussion, although at times auscultatory percussion aids in doing this by showing slight differences in the percussion note. The borders of the stomach, large and small intestine, can be determined only approximately, as will be explained later.

For percussion the posture of the patient is the same as described under Palpation. The fingers should always be used both as hammer and pleximeter. The strength of the percussion stroke should be regulated according to the nature of the organ percussed, whether solid or hollow, and its relation to the surface and to organs and tumors giving a different note. The percussion note is still further modified by the nature of the parietes, as the abdominal cavity is inclosed in part by the bony thorax and in part by the abdominal walls, varying in thickness and tension. As there is marked normal regional variation, it is necessary to be thoroughly familiar with the percussion sounds present in the usual anatomical divisions of the abdomen.

Left Hypochondrium.—This region includes that portion of the abdominal cavity lying beneath the ribs on the left side of the body. The vault of the diaphragm rises as high as the level of the fifth rib. The upper portion of this area is occupied by the thoracic viscera —heart and lungs, and by the abdominal organs—left lobe of the liver, stomach and spleen—and the percussion sound varies accordingly. Over that portion where the heart and liver are parietal the note is flat. Just below the area of flatness, toward the edge of the costal arch, the thin left lobe of the liver gives a superficial dullness on light percussion, while on forcible percussion a tympanitic quality can be obtained from the underlying stomach. To the left of the area of cardiac and liver flatness the lung is parietal from the fifth to the lower border of the sixth rib, and the sound is that of pulmonary resonance, with added tympanitic quality from the stomach. The proportion of the two sounds varies with the force of the percussion and the condition of the stomach and lung, pulmonary resonance increasing when the lung is overdistended, as in full inspiration or emphysematous dilatation; diminishing with retraction of the lung from any cause, and becoming flat when the complemental space of the pleura is filled by effusion. Distension of the stomach or displacement upward of this organ by increased abdominal pressure causes a relatively higher position of the diaphragm and corresponding increase in stomach resonance.

HALF-MOON SPACE (Traube).—This embraces all that portion of the lower thorax below a concentric line starting from the cardio-hepatic flatness in the sixth interspace, and extending downward to the anterior axillary line or mid-axillary line, which corresponds approximately to the lower border of the lung. In this space the stomach is parietal, and the note is high pitched and tympanitic, with a peculiar metallic, echo-like quality (stomach tympany).

The percussion note over this space may be altered by a number of conditions. (1) The upper boundary may be invaded by pulmonary resonance, due to increase in size of the lung, with depression of the diaphragm. (2) It may be flat from depression of the diaphragm by pleurisy with effusion. (3) The normal stomach tympany may be replaced by flatness, due to the filling of the organ with food or liquid. (4) Enlargement anteriorly of the left lobe of the liver from any cause may give an area of flatness or dullness. (5) Toward the axillary line in the region of the ninth to the eleventh rib enlargement of the spleen may give dullness or flatness. (6) Distension of the colon may give change in the tympanitic sound.

THE SPLENIC AREA.—This area is to the left of the half-moon space, and limits it in that direction. Although the spleen is contained in the abdominal cavity, with its long axis parallel to the tenth rib, and occupying the space between the ninth and the tenth ribs from the mid-axillary line in front to within 1½ to 2 inches of the vertebral column behind, under normal conditions but a small portion of its anterior border approaches the surface. On account of its respiratory and passive mobility, its position and corresponding effect on the percussion note will vary according to the posture of the patient. When lying on the back, it is displaced posteriorly; when lying on the right side, it is more anterior, but further removed from the surface. In the erect posture its position is more nearly horizontal, and slightly lower. It is impossible to accurately outline the normal spleen by percussion, surrounded and in part overlapped as it is by air-containing structures. The dullness obtained is only relative to the pulmonary resonance above and stomach tympany anteriorly, intestinal tympany below and flatness of lumbar muscles and kidney posteriorly.

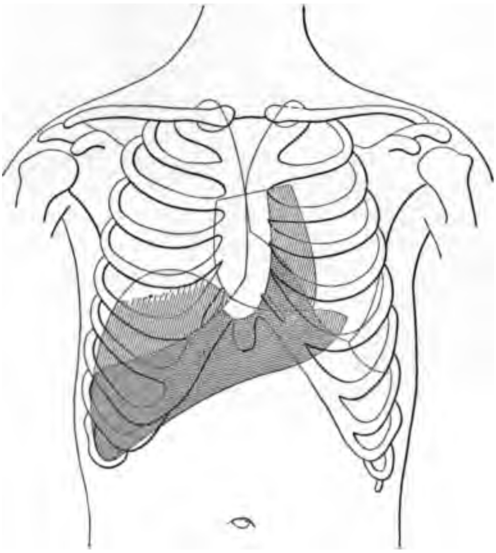
To determine the splenic area dullness, the percussion blow must vary according to the nature and condition of the overlying structures. Over the portion covered by the lung, beginning beyond the normal area, the percussion is first made toward the spleen with fairly strong blows until slight deep-seated relative dullness is detected. From this point the percussion proceeds with gradually diminishing force. From the gastric and intestinal borders only light percussion is used. In the normal condition of the lung, with stomach and intestine empty and not distended with gas, a small area of splenic dullness may be detected in the mid-axillary line or between it and the post-axillary line and between the lower edge of the ninth and upper edge of the eleventh ribs. Beyond this area the splenic dullness becomes progressively masked by increased pulmonary resonance as percussion proceeds toward the posterior border.

The size and position of the area of splenic dullness varies with the respiratory movements, being larger at the end of expiration and smaller or disappearing during inspiration. On account of the relation of the spleen to surrounding organs, the normal area of splenic dullness may be replaced *(a)* by increased resonance; *(b)* increased dullness or flatness. Increased resonance over the splenic area may be due to depression of the lower border of the left lung, dependent upon emphysema or to distension of the stomach and intestines, large and small, with gas (tympanites). Diffused dullness in the splenic area may be caused by consolidation of the lung overlapping the spleen, left pleural effusion or pleural thickening; fluid distending the stomach, or filling of the transverse portion or splenic flexure of the colon with fæces; also by general ascites. Absolute increase in splenic dullness occurs in all enlargements of the spleen, and when that organ becomes more parietal. On account of the effect of surrounding organs upon splenic dullness, the diagnosis of enlargement of the spleen should not be made by percussion alone, but the results should be corroborated by palpation.

Right Hypochondrium.—In the right hypochondrium all of that portion of the abdominal cavity that is covered anteriorly by the thorax is occupied by the liver, the upper border of which rises as high in the mammary line as the fifth rib, corresponding to the dome of the diaphragm.

Percussion over the liver gives two areas. The first corresponds with that portion which is separated from the thoracic wall by the lung—the area of relative dullness; and the second, that portion where the liver is parietal—the area of liver flatness.

FIG. 72.



Absolute and relative heart and liver dullness, also regional variation in thorax.

The pneumo-hepatic border of the liver corresponds to a line that separates the relative hepatic dullness from hepatic flatness. The position of this border and the area of hepatic flatness will vary according to the condition of the right lung, the border being lower and the area of hepatic flatness smaller with full inspiration and in emphysema, and the border being elevated with increase in the area of flatness in expiration and diminution of the size of the lung. The relative liver dullness cannot be detected over the entire upper portion of the liver, as the pulmonary tissue is too thick for the liver to be reached by the percussion vibration. (Fig. 72.)

The point at which the influence of the underlying liver upon pulmonary percussion will be detected will depend markedly upon the force of the percussion blow, which will also influence the determination of the pneumo-hepatic border. Proceeding from above downward, the percussion blows should be progressively lighter as the lower border of the lung is approached, where only the lightest stroke should be employed.

The pneumo-hepatic border should be determined by percussion from the area of relative dullness to absolute flatness, and also beginning over the flat area by percussing toward the area of relative dullness until faint resonance is detected. Normally the pneumo-hepatic border extends from the base of the ensiform cartilage in the median line to the upper border of the sixth rib in the mammary line, and to the eighth rib of the mid-axillary line. Well-marked liver dullness extends one or more interspaces higher, gradually shading off into pure pulmonary resonance.

The area of absolute flatness on the anterior portion of the chest extends from the pneumono-hepatic border to the free border of the ribs. Over the lower portion the thinness of the liver permits percussion vibration to reach the underlying air-containing organs, and gives a slight tympanitic quality to the note.

CONDITIONS MODIFYING THE AREA OF FLATNESS OVER THE LIVER.—The area of liver flatness may be enlarged upward by: 1. Changes in the thoracic organs due to *(a)* consolidation of that portion of the lung overlying the liver. *(b)* Retraction of the lung, causing greater portion of the liver to become parietal. *(c)* Effusion into the right pleural cavity or thickening of the pleura. 2. Displacement of the liver upward from any cause. In this condition the lower edge of the liver is also raised above the normal limit. 3. Absolute enlargement of the liver, which may be uniform or irregular, corresponding to the conditions noted under Palpation. In these conditions the enlargement of the liver upward is not so great as downward, except when secondary changes produce increase in the intra-abdominal pressure and displacement upward of the liver and diaphragm.

In irregular enlargements or tumors, the outline corresponds to the location of the tumors.

Extension of the area of flatness downward may be due to displacement from thoracic disease, causing descent of the diaphragm and liver. In this condition the liver may be forced down below the free edge of the ribs, and the area of absolute dullness is only relatively increased, the pneumono-hepatic border being also depressed. Absolute enlargement of the liver, uniform or irregular, causes the area of flatness to extend according to the change occurring in the liver.

Solid tumors or fluid extending to the liver cause the flatness to extend beyond the normal liver area.

Decrease in the area of hepatic flatness may be apparent or absolute. It is seemingly diminished whenever the area of normal flatness is encroached upon by distension of the lung above or by the stomach or intestine below. The greatest diminution in the size of the liver occurs in cirrhosis and in acute yellow atrophy.

The True Abdomen.—Percussion of all the anterior portion of the abdomen not inclosed by the thorax gives a tympanitic note, due to the underlying air-containing organs. In the epigastrium the stomach is overlapped by the left lobe of the liver, which is too thin to prevent the percussion vibration reaching the stomach. On light percussion, slight dullness due to the liver may be detected.

The percussion note obtained over the stomach, large and small intestines, is tympanitic in quality, and varies slightly in pitch over the different segments, according to the amount of air contained and the tension of the walls; but it is difficult to determine accurately the outline of the stomach and large and small intestines, on account of their varying size and degree of distension.

To determine the lower border of the stomach, it is examined when filled with fluid or artificially distended with air. When the patient is in the recumbent posture, the fluid gravitates to the most dependent portion; the note obtained over the viscus on percussion is tympanitic, and its lower limit is separated from the adjacent large intestine by a difference in pitch. The patient then assuming the upright position, the fluid—the amount of which may be increased by drinking water—causes the lower segment, which was previously tympanitic, to become flat on percussion.

Distension of the stomach by forcing air through a stomach tube, or by the production of carbon dioxide by having the patient drink separately a solution of tartaric acid (ʒss) and one of bicarbonate of soda (ʒss), causes the note to become markedly tympanitic and ringing. When it is not safe to cause forcible distension of the stomach, the large intestine may be distended by forcing gas from a Davidson syringe into the rectum, and so causing a difference in the pitch of the distended colon from that of the more relaxed stomach.

Conditions Modifying Abdominal Resonance.

Thickened Abdominal Walls.—Increased thickness of the abdominal walls, especially when due to fat, and also accumulations of fat in the omentum (omental lipoma), cause a slight diminution of resonance over the entire abdomen, which, however, is not sufficient to obliterate the tympanitic resonance of the stomach and intestines, but which masks the normal outline of the solid organs and renders the detection of tumors difficult, especially when they are small and not entirely parietal.

Fluid in the Abdominal Cavity, Ascites.—A small amount of fluid does not modify the percussion note over the abdomen, as the fluid gravitates to the most dependent portion, according to the posture. When sufficient in amount to rise above the symphysis in the erect position and above the heavy muscles of the flank in the recumbent posture, it gives a flat note where it is parietal, and, as the intestines are floated on the surface of the liquid, a tympanitic note is found above its level, which is always horizontal.

As the fluid moves freely in the abdominal cavity, the line of flatness and the area of tympany change with alteration of posture. As the fluid gives a flat note, like that of solid organs, it is always necessary to change the position of the patient in determining whether or not the extension of dullness beyond the normal limits is due to effusion into the abdominal cavity or to absolute increase in size of the solid organs.

When the abdominal cavity is extensively distended with fluid, or when the fluid is encapsulated, free movement may not occur. Although usually the intestines float above the fluid, adhesions or short mesentery may bind them down so as to prevent this.

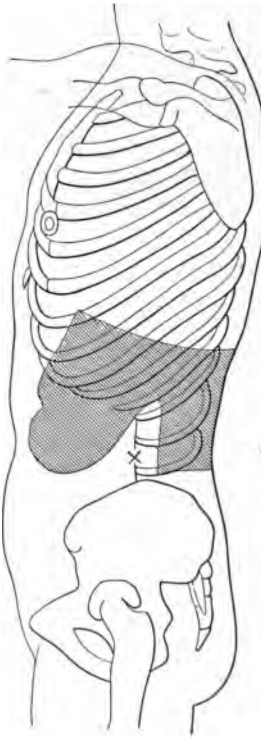
The relation of the area of tympanitic resonance to that of flatness is important in differential diagnosis of flatness due to ascites from abdominal tumors. In ascites, with the patient in the recumbent posture, the area of tympantic resonance occupies the centre of the abdomen around the umbilicus, changing position with that of the patient, and always resuming the uppermost position; while in solid tumors which are parietal the dullness occupies the most prominent portion of the abdomen, and is surrounded by tympanitic resonance, which does not change with altered posture.

ABDOMINAL TUMORS.—Abdominal tumors cause diminution in resonance according as they are parietal.

Tumors of the stomach and intestine are rarely large enough to give well-marked flatness, although when associated with thickened peritoneum there may be a slight diminution of resonance.

Fæcal accumulations in the large intestine may be detected by percussion, as they are usually parietal. They may easily be detected when they occupy the

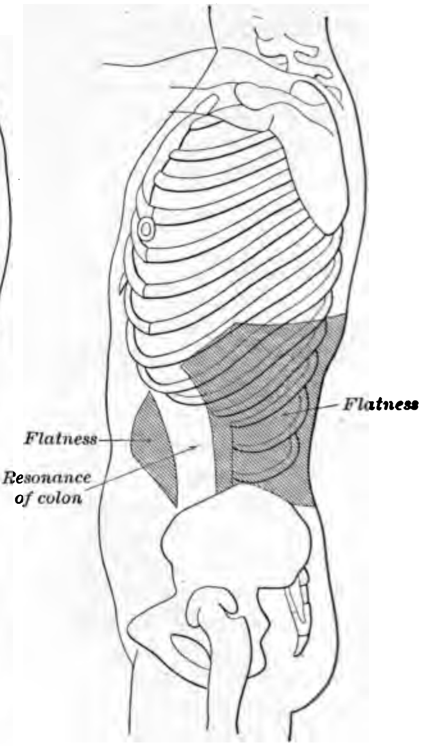
FIG. 73.



Tumor of spleen.

× Resonance between posterior margin and lumbar muscles.

FIG. 74.



Tumor of left kidney.

cæcum, the ascending colon or the sigmoid flexure. When situated in the hepatic or splenic flexures, they increase the areas of dullness of liver or spleen.

Enlargements of the liver and spleen, when they extend beyond the ribs into the abdominal cavity, give well-marked areas of flatness, sharply defined by the surrounding intestines. The enlarged organs are parietal, but it is only in exceptional conditions that a portion of the intestine intervenes between it and the abdominal wall. Tumors springing from the under surface of the liver, as hydatids, or irregular enlargements, as cancer, may have intestine in front.

Posteriorly the liver dullness does not extend to the spine, but well-marked splenic dullness may extend to the spine in the upper portion, while lower down there is an area of resonance between the tumor anteriorly and the edge of the lumbar muscles posteriorly. (Fig. 73.)

Tumors of the kidney are not parietal throughout, and the area of flatness is usually divided by the colon in front of it. Posteriorly the flatness extends to the spine throughout. (Fig. 74.)

In tumors of the pelvic organs the dullness extends upward from the pubic arch, and is surrounded by intestinal resonance in the flank and above. Dullness over the tumors may be diminished by distension of the intestine with gas (meteorism) or by air in the peritoneal cavity (perforative peritonitis).

When dullness in the flank is present, not extending also to the central zone of the abdomen, with resonance between the dullness and the central portion of the abdomen and the pelvic brim, the tumor is not of pelvic origin, but is from the deeper tissues (pancreas, retroperitoneal glands, etc.).

CHAPTER XVIII.

AUSCULTATION.

Auscultation of the abdominal organs gives very little aid in differential diagnosis, and is limited to *(a)* splashing sounds made in hollow organs containing both air and fluid; *(b)* gurgling, churning or cooing sounds, due to passage of fluid through the intestinal canal; *(c)* friction sounds, produced by the roughened peritoneum over solid or hollow organs which move with respiration; *(d)* vascular or hæmic sounds.

(a) Splashing sounds (succussion) may be heard over the stomach and large intestines when they are agitated with short, sharp blows with the tips of the fingers.

In the stomach this occurs when the viscus contains both air and fluid and is to a certain degree relaxed. It was formerly considered diagnostic of gastroectasis, but can occur without any pathological condition of the stomach being present. Its chief value as a sign of enlarged stomach is in finding it persistently below the normal limit of the stomach.

Over the cæcum, on account of the liquid character of its contents, succussion sounds may also be produced. When found beyond the head of the cæcum, especially in the transverse portion of the colon, it indicates an abnormally liquid state of the fæcal mass in these locations. In the small intestine succussion cannot be induced.

(b) Gurgling or churning sounds are heard over the stomach and intestines, due to the passage of liquids from the narrowed portions into the wider areas beyond. Over the cardia, normally, a hissing murmur is heard soon after swallowing liquids. In obstruction at the

cardia from any cause, the normal deglutition sound may be replaced by a gurgling or rushing sound occurring at a relatively later period, and due to the forcing of the contents of the œsophagus through the obstruction.

Over the small and large intestines, normally, cooing, gurgling sounds are heard. When the intestines are distended with gas and the peristaltic activity is increased, this may become loud enough to be heard at some distance from the patient.

In tympanites with lessened peristalsis, as occurs in paralysis of the intestines, and in peritonitis, absence of this normal sound in proportion to the distension is of diagnostic importance.

Over the ileo-cæcal valve, gurgling can frequently be heard on firm pressure. This has been frequently cited as one of the symptoms of typhoid fever, but it is not pathognomonic of this disease, as it occurs whenever the contents of the small intestine are more liquid than normal and when there is slight distension of the ileum with gas.

(c) Friction sounds are heard whenever the peritoneal coats are covered with inflammatory exudate and there is free movement between the surfaces. This is most marked in perihepatitis and perisplenitis, provided the normal respiratory mobility of the liver and spleen is not interfered with. It is rarely heard over hollow organs, although in acute localized inflammation of the stomach it may be present. The quality of the sound will vary according to the character of the exudation. In fibrinous exudation it occurs as fine, crackling râles. When adhesions are present, creaking, crumpling friction sounds may be heard.

(d) Vascular or hæmic murmurs may be heard over different portions of the abdomen. Solid tumors and growths pressing on the aorta or large blood-vessels may cause sufficient narrowing to induce a murmur, which is conveyed by the tumor to the surface, and is heard as a bruit, synchronous with the first sound of the heart,

or it may be a little delayed. Aneurismal dilatation of the aorta or abdominal vessels may cause a similar murmur to be heard. They are differentiated from pressure murmurs by the presence of expansile pulsations.

Over the liver a systolic murmur may be heard, due to the conveyance of tricuspid regurgitant murmur..

In tumors of the uterus, arterial sounds may be present. In pregnancy, in addition to the uterine souffle, there is heard the fœtal heart beat. Hæmic sounds, simulating the uterine souffle, may also be heard in extra-uterine pregnancy and in vascular tumors of the uterus, but the fœtal heart sound is not heard..

PART V.

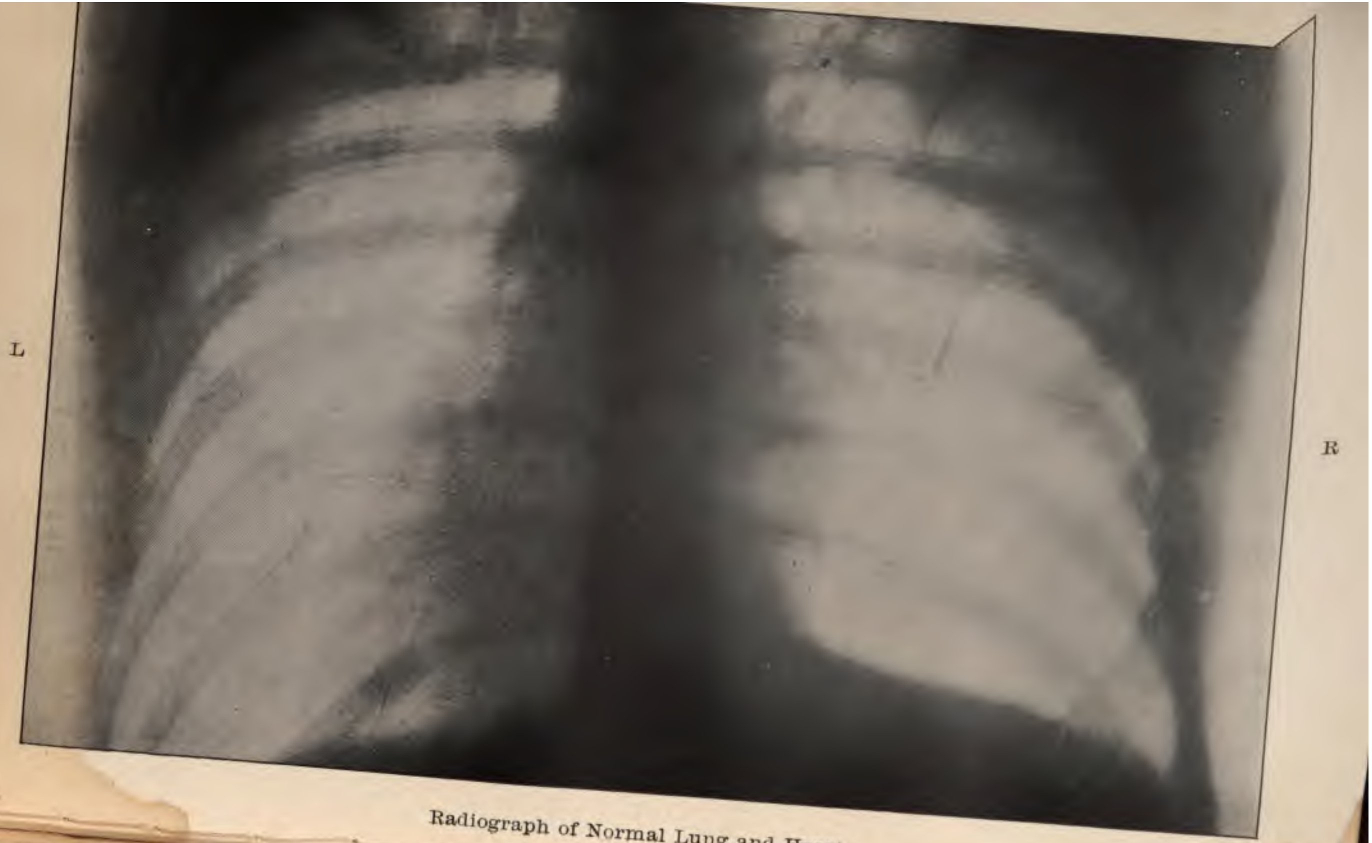
EXAMINATION WITH X RAY.

For making X-ray examination of the body, it is necessary to have apparatus of great power, and also means for adjusting the quality of the ray emitted by the tube, which may be accomplished either by inserting a series of spark caps in the circuit connecting the tube or by adjusting the vacuum of the tube. There are on the market a number of tubes provided with vacuum regulating devices, all of which depend for their action upon the liberation of a little gas from some salt which is contained in a small chamber connected with the tube.

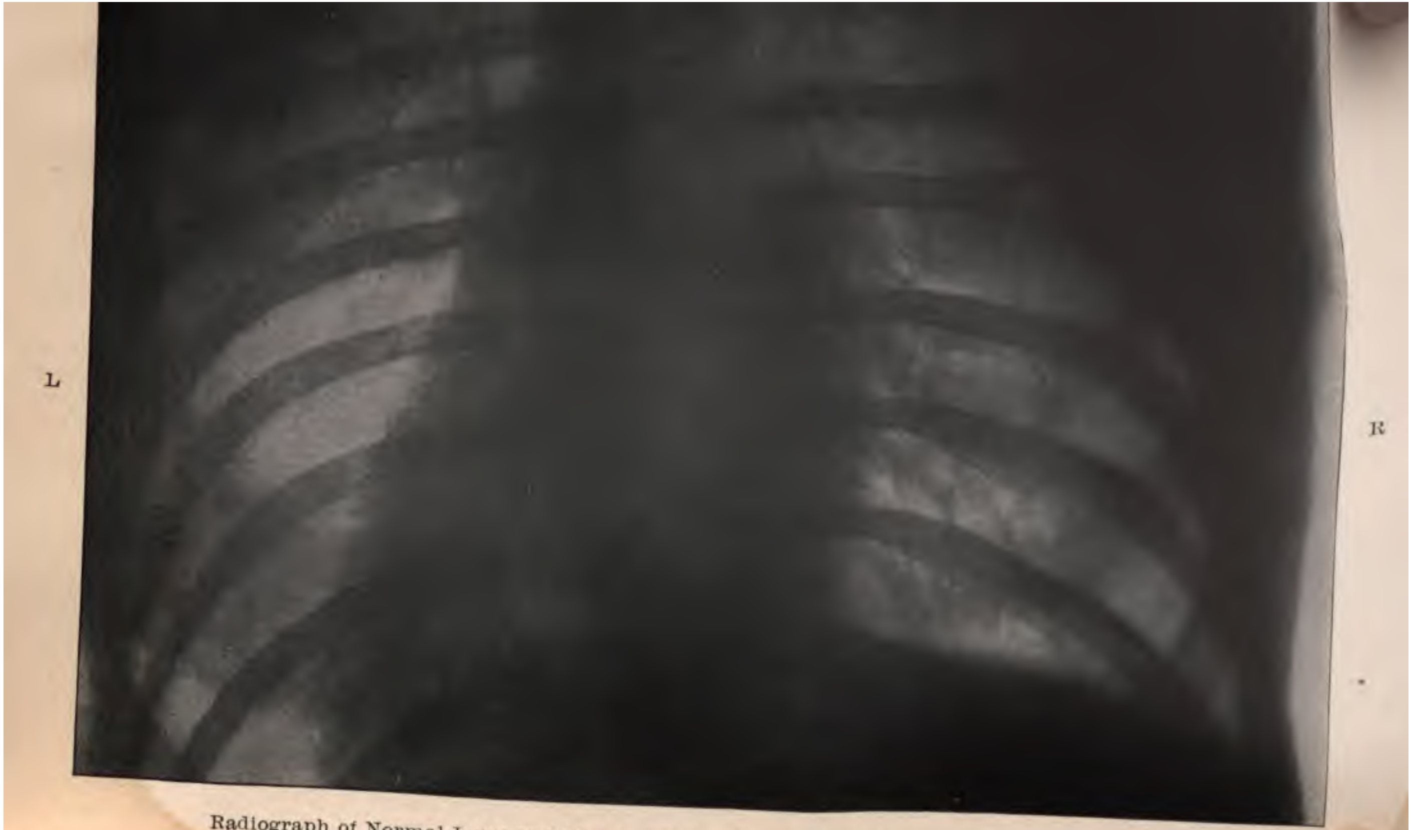
The exciting apparatus maybe either a static machine, an ordinary induction coil, or a coil of high frequency type. If an induction coil is used, it should be capable of delivering heavy sparks, 10 or 15 inches long, and should be provided with means for giving very rapid interruptions of the primary circuit. The turbine mercury interrupters and the liquid or electrolytic interruptions are available for this purpose. The induction coil has the advantage that it occupies a comparatively small space, and its action is not affected by the weather. If the static machine is used, it should be of a rather large size, having, for example, 10 to 16 revolving plates of about 30 inches diameter. The static machine gives a very steady excitation of the Crookes tube, and is therefore very satisfactory for fluoroscopic examinations. It is, however, more or less susceptible to changes in weather conditions.

On account of the constant movements of respiration, the fluoroscope examination of the thoracic viscera is perhaps more satisfactory than the radiograph. Much valuable information may be obtained by observing the movements of the parts, as, for example, the excursions of the diaphragm, the pulsations of the heart or of aneurism, the change of level of fluid in the pleural cavity, due to change of position of the subject, etc. The facility with which these examinations can be made and the accuracy of their findings will vary with different subjects. Obviously, a small, thin subject will give a more satisfactory fluoroscopic picture than a large, muscular one. At first it may seem that by increasing the intensity and penetrating power of the rays we could examine a large subject as well as a small one, but this is not the case. It is easy enough to produce X rays of sufficient intensity to penetrate any human body, but in order to make a satisfactory fluoroscopic examination or radiograph it is necessary that these rays be intercepted to varying extents by the different structures under examination, so that they will cast shadows of discriminable density. A thick layer of muscular or adipose tissue, although it may transmit the ray, to a certain extent confuses the shadow of substances of less capacity which lie beneath it; hence it is impossible to differentiate the tissues under examination by increasing the power of the X rays.

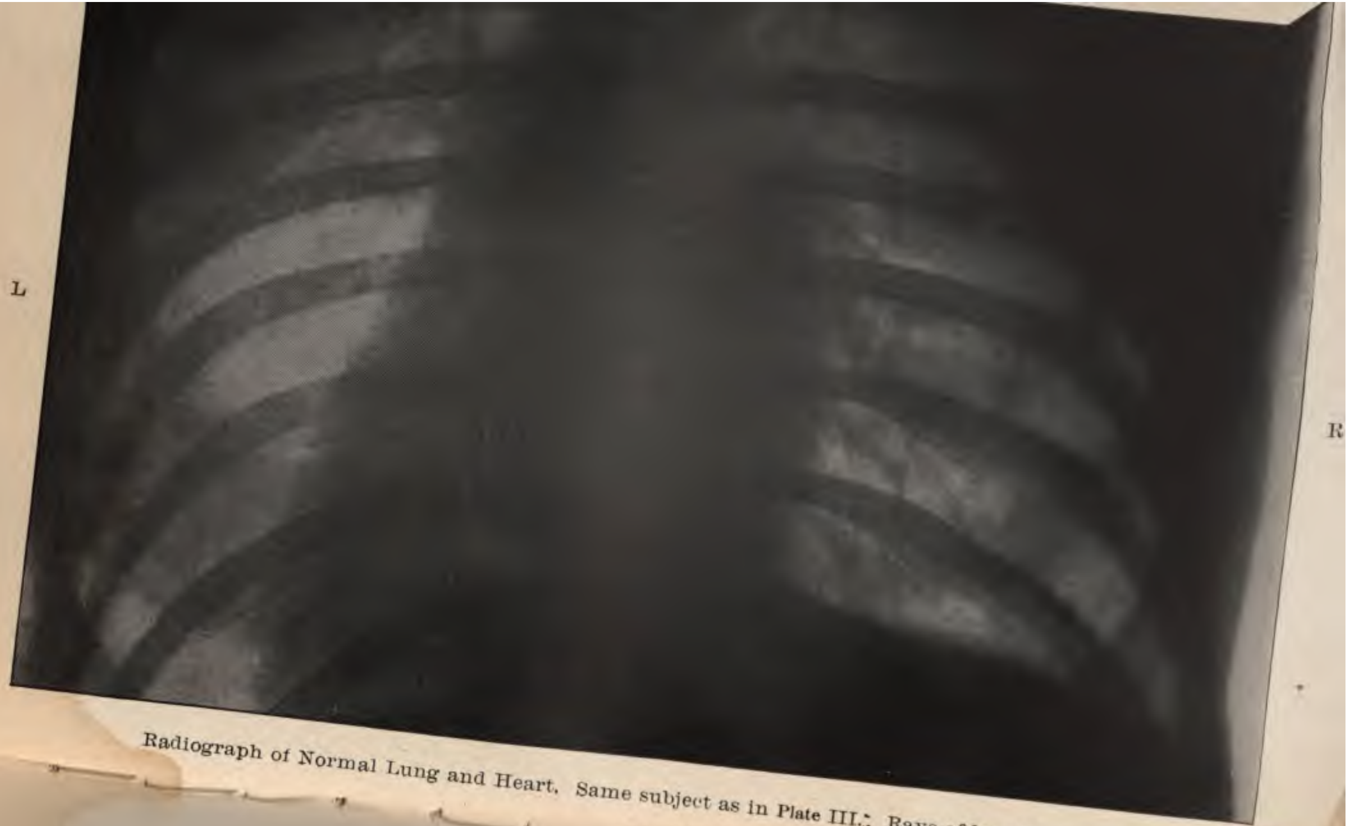
Fluoroscopic examinations should be made in a darkened room. It will be found that it takes the average eye from five to fifteen minutes to adjust itself so as to use the fluoroscope to the best advantage. All clothing should be removed from the patient, and the examinations can be conveniently made with the patient in a sitting position, with the arms resting over the head, so as to draw the scapulæ away from the median line. It is well to have two fluoroscopes—a large one, which will show the entire region, and a small one, with which small parts may be examined and the light from a large



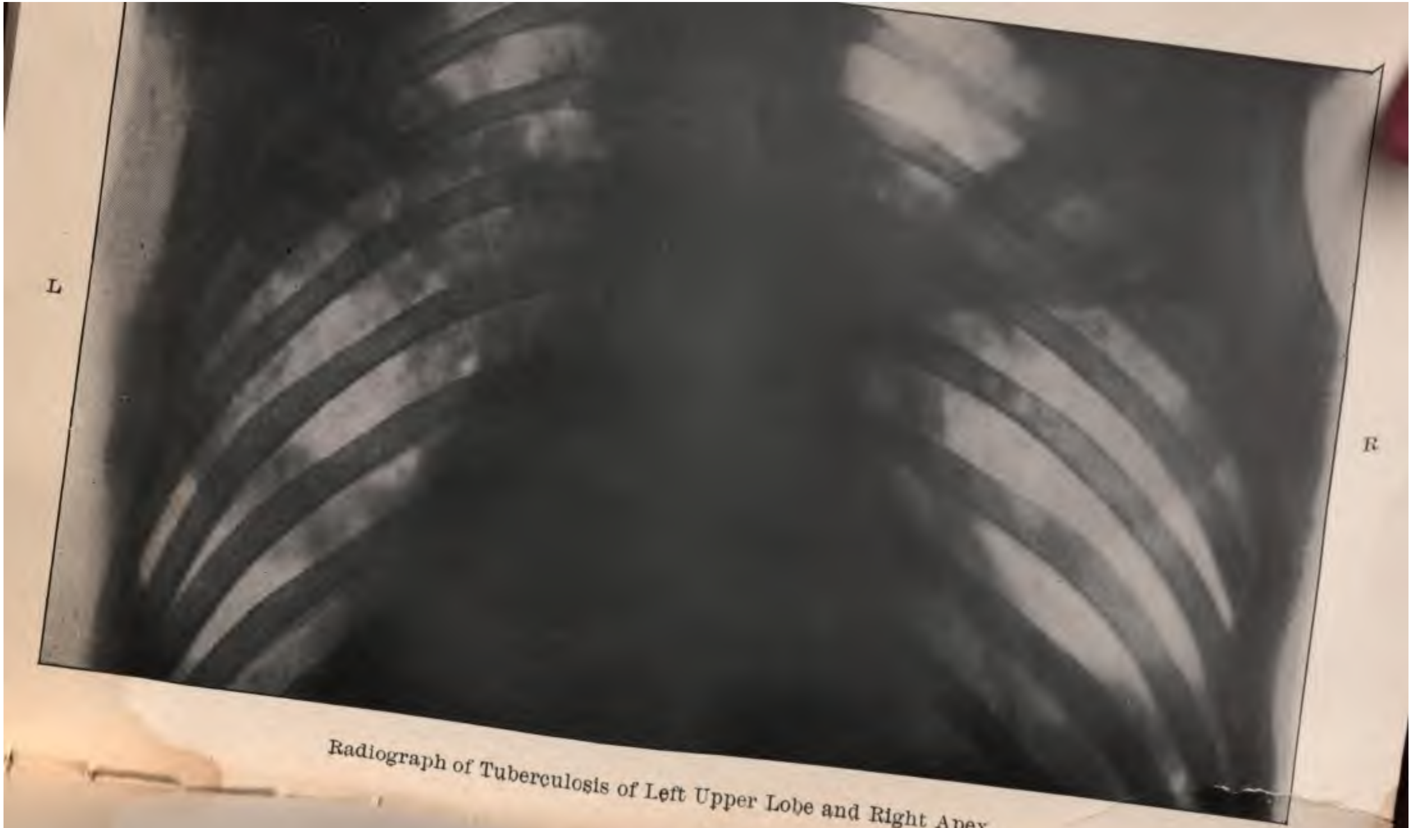
Radiograph of Normal Lung and Heart.



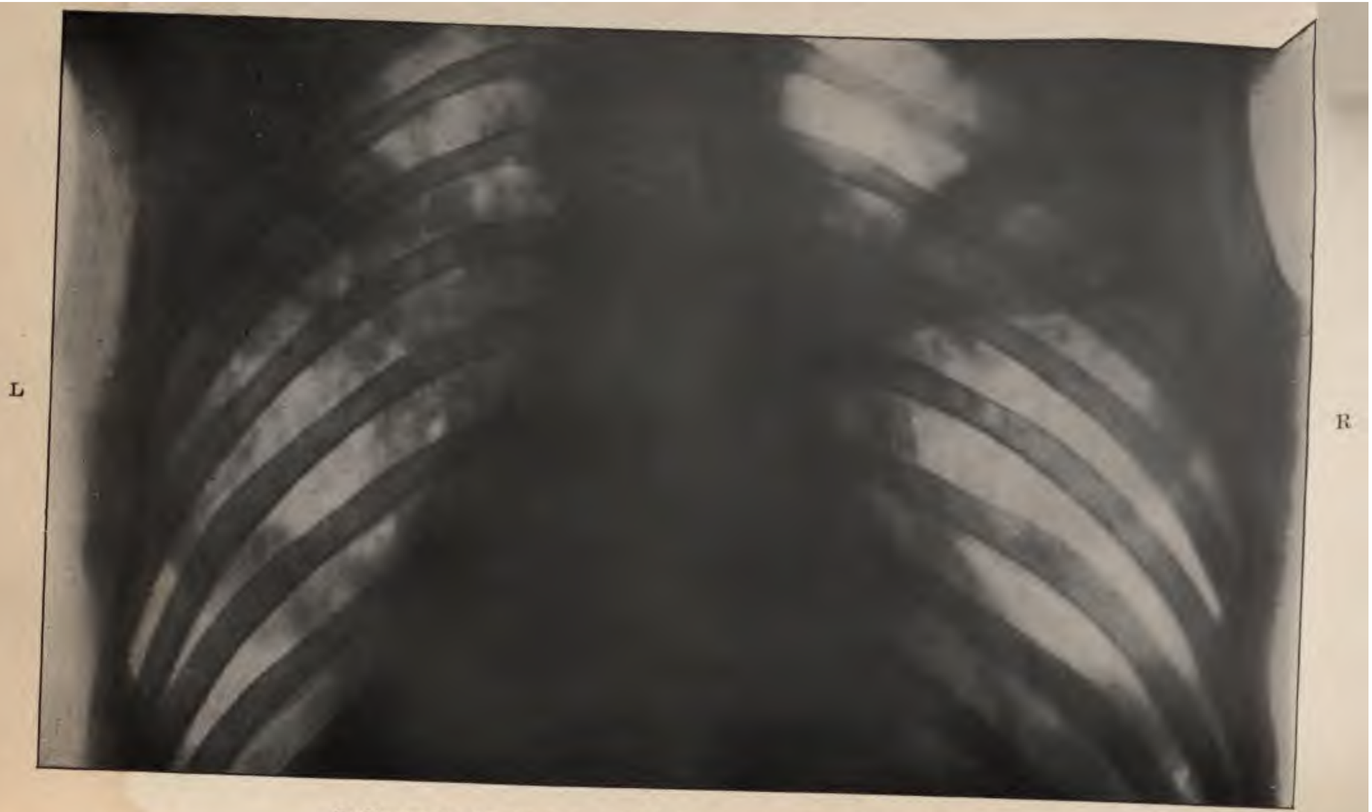
Radiograph of Normal Lung and Heart. Same subject as in Plate III.; Rays of lower power.



Radiograph of Normal Lung and Heart. Same subject as in Plate III. Rays of lower power.



Radiograph of Tuberculosis of Left Upper Lobe and Right Apex.



Radiograph of Tuberculosis of Left Upper Lobe and Right Apex.

PLATE VII.

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area excluded. Sometimes it will be convenient to examine the patient in a recumbent position. In this case it is well to place the subject on a stretcher, placing the tube underneath and the fluoroscope above. The canvas of the stretcher offers very little obstruction to the X ray. A protective screen of thin aluminum is sometimes interposed between the X-ray tube and the subject under examination, to prevent any burning by the rays. This screen offers very little obstruction to the X ray, and therefore interferes very little with the examination.

For the reasons pointed out above, the radiograph of the thorax will usually not be so satisfactory as the fluoroscopic examination, though it may be valuable as a permanent record. The apparatus required for making the radiographs will be practically the same as those described for fluoroscopic examinations, with the addition of necessary plates, etc.

Observation by means of the X ray of the thorax and abdomen presents many points of interest to the clinician. A thorough knowledge of the normal appearance of the different parts of the body under the X ray is absolutely necessary for the proper use of the fluoroscope, and for correct interpretation of the skiagraphs as a means of diagnosis, otherwise false interpretation will be made. The use of the X ray in medicine should be accompanied by the use of other methods of diagnosis, and should never be relied upon alone.

For the beginner, the subject to be examined should be carefully selected. A young person, preferably an adult male about twenty years old, and not too fleshy, is the best. It should be constantly borne in mind that each subject will present a picture that has certain individual peculiarities, according to the thickness of the soft parts, the bony structures and antero-posterior diameter of the chest. The fluoroscope has many advantages over the skiagraph for a beginner, as the outlines are more distinct, and it is possible, by varying the intensity of the light, to obtain a better view of the thoracic and abdominal organs and to determine their outlines.

The Normal Picture.—As in other methods of diagnosis, the examination should be systematically conducted. At first a general inspection of the thorax from above downward is made. The picture upon the screen shows in the median line corresponding to the sternum, spinal column and mediastinal spaces, a dark shadow at the level of the third rib, which widens out into a more or less spherical figure, corresponding to the ventricles, and resting on the dome of the diaphragm, which appears as a dark line. On either side of this central shadow appears a light field, corresponding to the pulmonary tissue, which should present a thin, foggy, uniform shadow, varying during the phases of the respiratory act, being lighter on full inspiration and darker at the end of expiration, according to the rarefaction of the lung. Traversing the light area of pulmonary tissue, the ribs are noted, their prominence varying with the intensity of the light used. Beyond the thorax, the bony structures and soft parts are readily distinguishable. (Plates III and IV.)

In addition to the outline of the organs above mentioned, the extent of their motion should also be noted. Ventricular contraction is well defined, also slight pulsation of the aorta can be seen. The line of the diaphragm varies with the respiratory act, descending with inspiration, when the complemental spaces of the pleura become distinguishable as a transparent area. The movements of the diaphragm should be uniform, both sides acting simultaneously and to the same extent. With the descent of the diaphragm and rarefaction of the lung, the cardiac position changes and the outline becomes more distinct.

The Effect of Changes in the Lung on the Fluoroscopic Picture.—These may cause the shadows to be more tense and darker when the density of the lung is changed by increase in the normal structure or by filling of the alveolar spaces; or the opposite when rarefaction of the lung occurs from increased size of the alveolar spaces, or when there is destruction of tissue with cavity formation. They may also cause change in the movements of the diaphragm and in the position of the heart and mediastinal structures.

CONDITIONS CAUSING DARK AREAS.—Tuberculosis. It should be borne in mind that the right apex is not so markedly transparent as the left. This corresponds to the normal variation noted under palpation, percussion and auscultation. The amount of darkening will correspond to the degree of change. (Plates V and VI.)

As in other methods of examination, the fluoroscopic picture of the two sides is comparative, and should always be judged on this basis. When the tubercular area is central and surrounded by emphysematous lung, the shadow of the affected portion is more pronounced.

In the incipient cases a slight denseness in the shadow will be observed in one apex when carefully considered in comparison with clearness of the normal shadow, and with the apex on the other side. As the pathological process extends until consolidation or cavity results, the shadow will progressively become deeper and larger until the change on the two sides is very marked. The fibroid changes are evident by the absence of the normal foggy shadow and by a more dense shadow over the areas involved. (Plate VII.) This condition is often noticeable in the lower portion of the chest, secondary to the diaphragmatic involvement. Retraction and thickening of the pleura give a darker shadow, and marked loss of motion over the affected area. (Plate VIII.)

Pneumonic consolidation. In lobular pneumonia the dark areas correspond to the site of the lesion, and have the same general appearance as those of tubercular infiltration. Croupous pneumonia gives a well-defined shadow, corresponding to the degree of consolidation and the division of the lobes. Radioscopy aids greatly in differentiating pneumonia from primary or complicating effusion, showing the relation of the lung to the diaphragm. In pneumonia, on full inspiration, the descent of the diaphragm shows a light line between the lung and the moving shadow of the diaphragm. When effusion occurs, primary or secondary, this line is not seen. Pleural effusions cause a uniform dark shadow. The character of the fluid does not influence this shadow. In addition to the shadow caused by the fluid, it is possible to determine the line that the fluid assumes in the chest and the amount of displacement of the thoracic viscera.

Calcareous deposits in the lung or fibroid thickening also cause slight shadows. The bronchi may give an ill-defined shadow on either side of the median dark space, and must always be considered. Enlargements of the glands cause shadows to appear.

CONDITIONS CAUSING LIGHTER SHADOWS.—The lung transmits light more readily in emphysema, and there is less change of shadow during respiration. The movements of the diaphragm are not as great; the heart appears unusually clear, and also assumes the typical position. In pneumo- and hydro-pneumothorax the shadow is less marked over that portion of the pleura which contain air (Plate IX), while when fluid is present a dark shadow is seen below its level. The upper level assumes a horizontal line, in contradistinction to the curved line seen in simple effusion. In hydropneumothorax the splashing of the fluid is plainly seen on shaking the patient, and also the change of level on his changing position. The pulmonary shadow is present above the level of the fluid, and is slightly deeper in density, owing to the compression. In pneumothorax the lack of motion of the diaphragm during inspiration is the most striking feature. The displacement of the heart and the relatively denser shadow of the opposite

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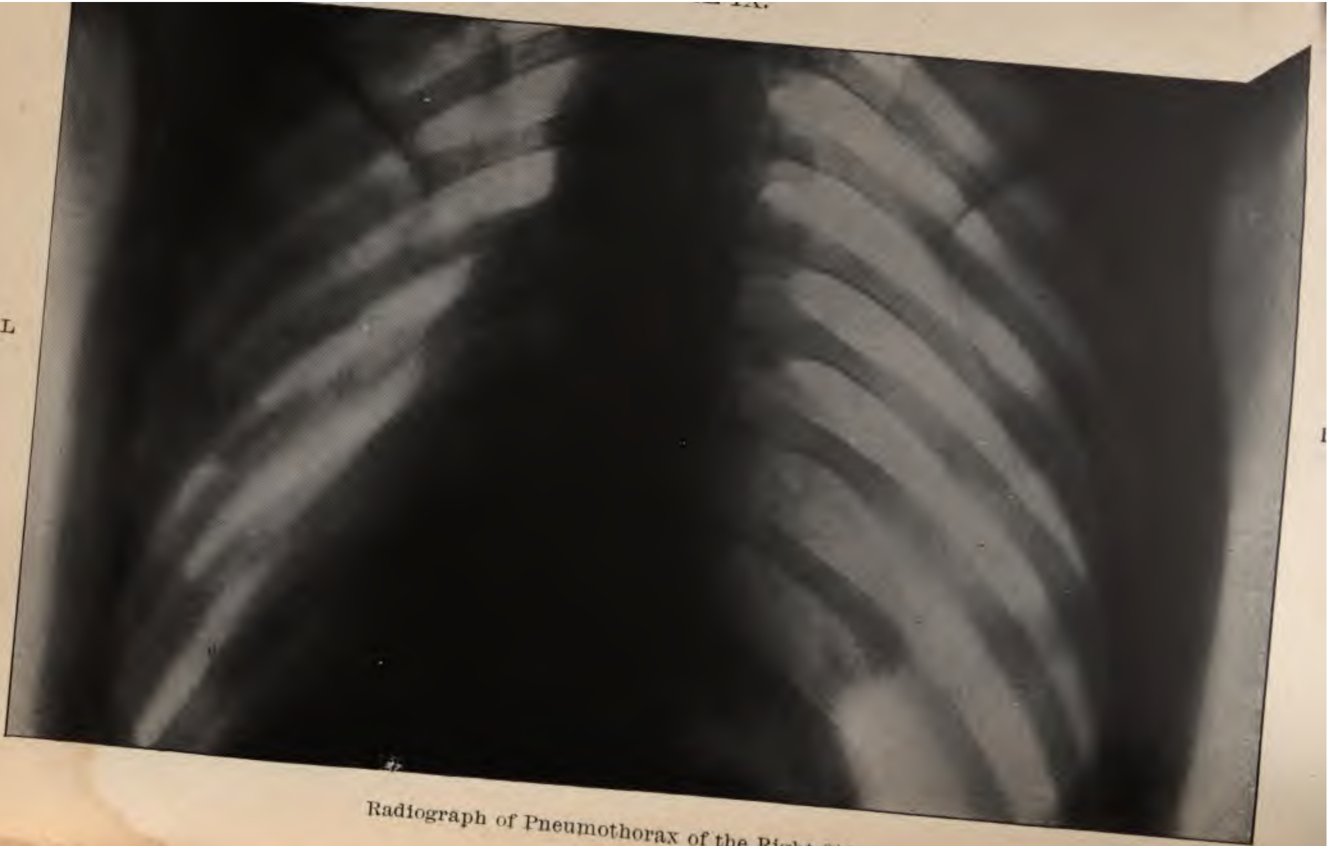
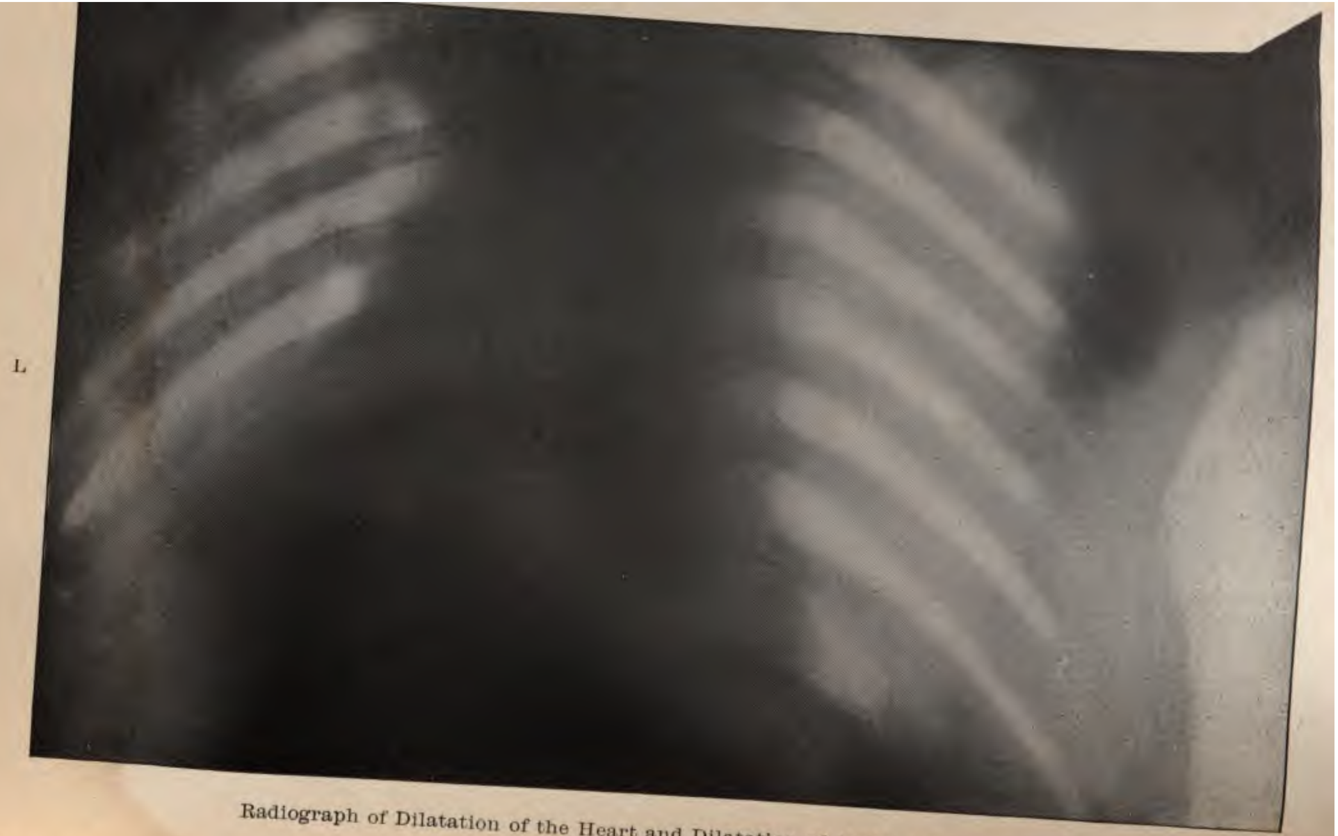
IX.



Radiograph of Pneumothorax of the Right Side.



Radiograph of Dilatation of the Heart and Dilatation of Aorta. Same case as Fig. 66.

tain calcareous deposit, or the cavity is surrounded by consolidated lung, a shadow may replace the light area.

Examination of the Circulatory System.—For satisfactory examination of the borders of the heart, the lung and pleura must be clear. Normally a dark shadow is caused by the ventricles, while the right auricle causes a faint shadow to be seen just to the right, and on the left a fainter shadow is caused by the left auricle. The rarefaction of the lung causes these shadows produced by the auricles to become clearer, so that they are more pronounced at the end of full inspiration than in expiration, and also in emphysematous dilatation than in normal lung. Dilatation of the cavities or distension by blood causes a correspondingly darker shadow. As has been mentioned, the intensity of the shadows will vary according to the amount of light used. The pulsations of the heart and aorta may be seen, especially during inspiration.

The exact cardiac outline can be mapped out on the chest by means of an indelible pencil; the lower end encircled in the metal cylinder. After carefully outlining the heart and locating the apex, the tracing can be made direct from the chest wall. The tracing should include the nipple and one or two bony parts of the chest. This chart can be filed and retained as a permanent record. (See Fig. 66.)

Cardiac Diseases.—The fluoroscope, in selected cardiac cases, will materially assist in determining the cardiac area, shape and relation to the surrounding viscera. In the following diseases important points will be enumerated in detail:

PERICARDITIS WITH EFFUSION.—The fluid in pericarditis changes the shape of the cardiac shadow, giving the broad base below; the pulsating apex is less visible. A point of great value in this condition can be noted on deep inspiration; as the diaphragm descends the pericardium can be seen to pull away from the cardiac mass, and is distinguished by the slight change in density between the pericardial fluid and the cardiac wall.

ADHESIVE PERICARDITIS.—The displacements due to retraction are visible, and also those which occur when the pericardium is displaced by fibroid changes in the lung.

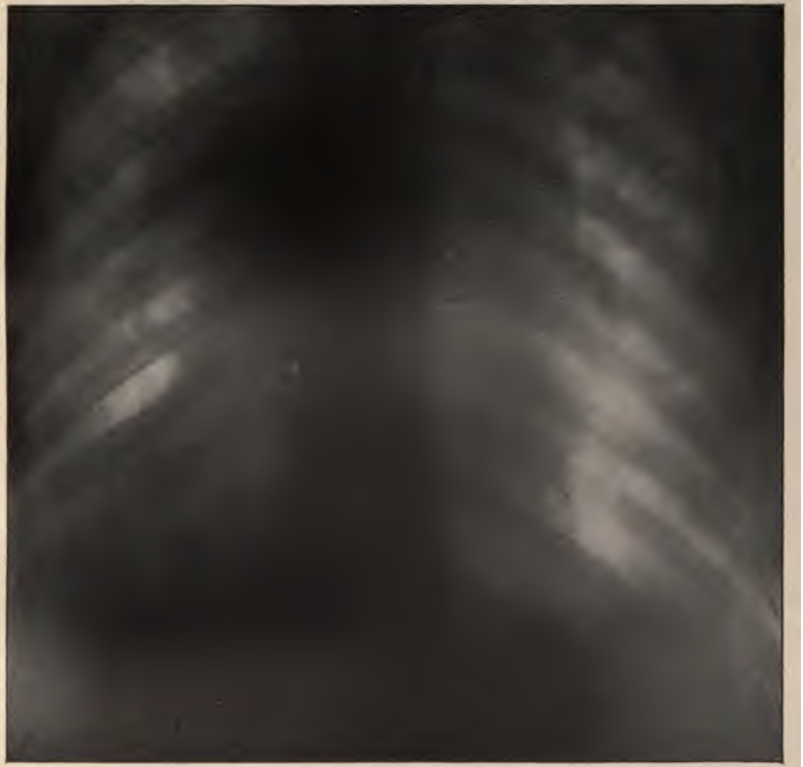
HYPERTROPHY AND DILATATION.—Changes in size and in the shape of the cardiac shadow can be, by means of the scheme outlined above, readily observed and recorded accurately for future observation. (Plate X.)

MITRAL STENOSIS.—The change in the shape of the heart in this condition is very noticeable. The axis of the shadow is changed; the apex is carried upward and to the left, and the long diameter is almost parallel with the diaphragm.

ANEURISM.—The localization of aneurism at the base of the heart, or involving the arch of the aorta, is probably one of the most important results of fluoroscopic work. The bulging of the vascular wall can be noted accurately, and its relation to the base of the heart or great vessels plainly demonstrated. (Plates XI and XII.)

The Radiograph in Cardiac Diseases.—The radiograph in cardiac diseases has been of much less assistance than the fluoroscope, yet, where available, it places in our hands an absolute picture of the conditions under consideration. In many subjects a radiograph can be obtained which will determine the outline and position of the heart, location and size of the aneurisms of different portions of the aorta.

PLATE XII.



L

Radiograph of Aneurism of Ascending Portion of Thoracic Aorta.

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